Kasr Al Aini Faculty of Medicine,
Cairo University

Department of Pediatrics

Pediatric Emergency & Critical Care division

Resident Pre-ICU guide

In life, as in critical care;
improvement is generally gradual,
sudden events are usually bad ones,
supportive care is very important
and the good news is "you're still alive"

Critical Care Medicine:

A multidisciplinary medical and nursing field that deals with patients who have, or are at risk of developing, single or multiple organ/ system failure as a result of illness or injury.

The ICU:

- **The unit that deals with critically ill patients**
- **The system, team, equipment and processes; not just the place**
- **"Intensive care is the people who care intensively" (Prof. M. El-Naggar)**
- **Multidisciplinary management is important. That said, all supportive management of critically ill children is within your scope. Your patient is primarily your responsibility. Comanagement cannot mean that anyone else is managing your patient INSTEAD OF YOU.**
- **Doing the right thing at the wrong time can be useless**

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EMERGENCY CONCEPTS

EMERGENCY MEDICINE is the field which deals with acute life-threatening conditions and aims to save lives through prompt and appropriate supportive and specific management.

System failure and system support:

- **Any serious illness can lead to system failure**
- **Multiple organ system failure (MOSF) can occur:**
 - In sepsis, serious trauma and poisoning
 - Following cardiopulmonary resuscitation
 - Following single system failure
- **All systems interact with eachother in health and disease**
- **Emergency medicine addresses all systems at the same time**
- **Recognition and support of failing systems is generally more important and more urgent than full diagnosis and specific treatment**
- **There are generally three levels of care:**
 - ***Emergency departments:*** receive patients initially and manage cases requiring less than 24hrs of hospital care
 - ***Intermediate care units:*** manage moderately serious cases needing more than 24hrs. They may receive cases from emergency departments or from intensive care units (after improvement)
 - ***Intensive care units:*** receive the most serious cases

Triage:

The process of classifying or sorting patients according to the priority (degree of urgency) and needs of their condition

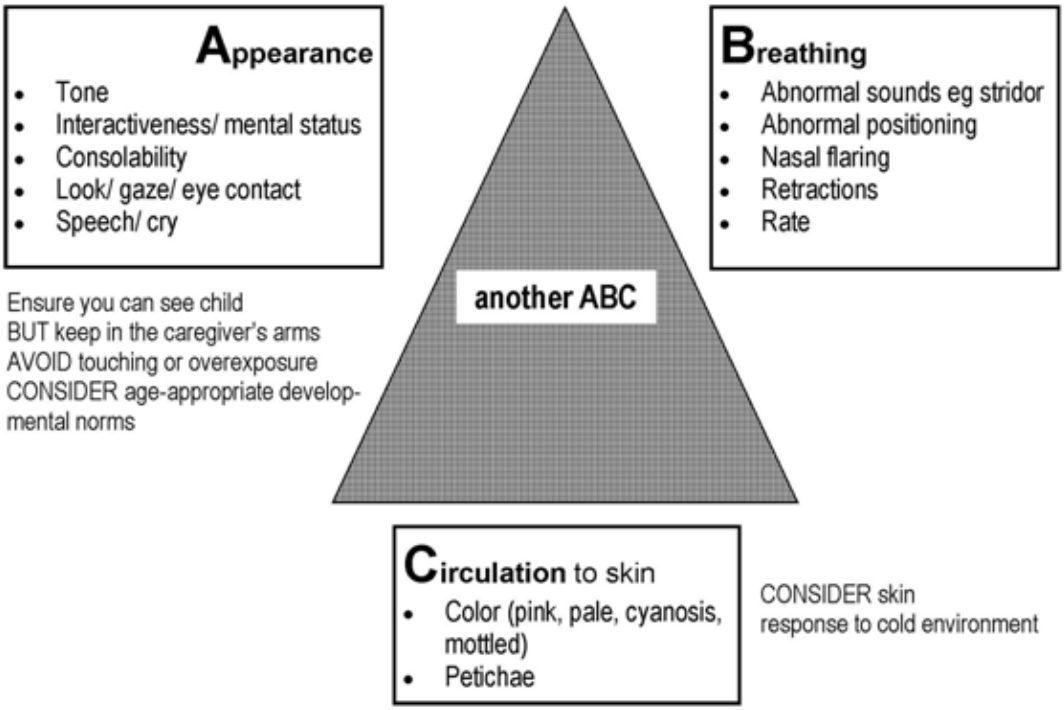
- It is used in busy emergency departments as the most urgent cases should not wait and many less urgent cases may be present. It is also used in wars and mass casualties.
- **Patients are generally rapidly classified into 3 levels:**
 - *Those with immediate resuscitation needs*
 - *Urgent cases with priority for assessment and management*
 - *Less urgent or non-urgent cases*

EMERGENCY APPROACH

5 successive steps should be done rapidly; replacing the traditional medical steps:
Primary survey ➔CPR ➔ Secondary (system) survey ➔ Definitive care ➔ Monitoring

1. **Primary survey ABCDE ;** ***to determine the immediate need for resuscitation***
 - **A: Airway:** patent or not
 - **B: Breathing:** breathing or not
 - **C: Circulation:** heart is beating or not
 - **D: Disability:** patient is conscious or not
 - **E: Exposure:** Uncover the patient to look for injuries or bleeding
2. **Cardiopulmonary resuscitation (CPR):**
3. **Secondary survey** To identify system failures
4. **Definitive care**
 - Supportive treatment to prevent and treat system failures
 - Specific treatment of the causative disease
5. **Monitoring**
 - Continuous monitoring using devices (pulse oximeter, ECG, HR, RR, BP, etc)
 - Clinical monitoring
 - Investigations: Recording of laboratory and radiological changes
 - Therapeutic monitoring: response to therapy, new problems and changes in management

PEDIATRIC ASSESSMENT TRIANGLE



INTERPRETATION			
A	**B**	**C**	**Conclusion**
ABNORMAL			Neurological problem
	ABNORMAL		Respiratory distress
ABNORMAL	ABNORMAL		Respiratory failure
		ABNORMAL	Compensated shock
ABNORMAL		ABNORMAL	Decompensated shock
ABNORMAL	ABNORMAL	ABNORMAL	Cardiopulmonary failure

Pediatric assessment triangle (PAT) is a simple and rapid triage tool for identification of sick children and classification of the probable illness category. PAT uses an assessment of general condition (appearance), breathing and circulation; based on rapid inspection.

EMERGENCY TRIAGE & MANAGEMENT SEQUENCE

TRIAGE SEQUENCE 123		SIGNS			ACTION
1st Check ABC		Appearance	Breathing	Circulation	
	EMERGENCY SIGNS (Immediately life-threatening condition)	- Coma[1] - Convulsions now[2]	- Airway obstruction - Central cyanosis[3] - Severe RD[4]	Shock	IMMEDIATE stabilization/ resuscitation
	OR				
	PRIORITY SIGNS (Should NOT wait)	Lethargy, Marked irritability	RD (grade II+) or dyspnea at rest	- Severe dehydration - Severe pallor - Active bleeding	URGENT assessment and initiation of management
2nd Is it a HIGH-RISK SITUATION?					
	PRIORITY SIGNS (Should NOT wait)	High risk situation: - Not able to drink/ breastfeed, vomits everything[5] - Convulsions this illness[6], neck rigidity - Serious trauma/ poisoning/ suspected surgical emergency - Urgent referral note			
3rd Non-Urgent; No emergency or priority signs Are there signs of concern?					
	SIGNS OF CONCERN	- Age <2 Mo - Known chronic illness - No improvement on previous treatment for this illness - Severe malnutrition - A condition you are not familiar with			CAUTIOUS assessment; don't take lightly
NO SIGNS OF CONCERN		None of the above			ROUTINE assessment

1 Coma does NOT include lethargy or disorientation but includes stupor
2 Patient having ONGOING fit, not previous fits
3 Excluding previously diagnosed cyanotic congenital heart disease
4 Severe RD= severe tachypnea, marked retractions, or grunting
5 Inability to eat is NOT by itself a high risk situation. Vomiting everything must be verified by a small amount of oral clear liquid or breastfeed. Do NOT just rely on history
6 Present, not past, history highly suggestive of a seizure

CAUTION:
Triage result determines priority of assessment; NOT its result
While most non-urgent cases can wait to be assessed, they may still need admission

- Do NOT wait for ANY further assessment
- Immediately initiate resuscitation/ stabilization
- Do NOT leave patient unattended until stable
- WHEN STABLE: proceed with assessment as with priority cases
- IF NOT STABILIZED: strongly consider ICU
- Do NOT keep patient in resuscitation area as it might be needed by other patients

- Should not wait to be assessed
- Assess to determine needs; many will require hospitalization or emergency short-stay, while few might need ICU
- Give priority in management once needs are known

5- POINT DISCHARGE CHECK

1- No emergency needs
2- All care documented
3- All tests reported/ reception arranged for

4- Discharge orders
- Drugs
- Feeding
- Fluids
- Observation
- Cautions

5- When to return?
- Routine follow-up (when? Where? for what?)
- Conditional (if not improved after——-)
- Urgent (danger signs)

Assessment of needs

Class	Description	Action
A	Possible need for ICU	• Keep in critical zone of ER • Many may not need ICU with proper care • Arrange transfer to ICU if & when needed & possible
B	Needs admission, ICU unlikely	• Admit directly to in-patient department • Keeping patient in ER is a waste of time & ER resources
C	Needs short stay in ER (<24 hrs)	Admit to ER (on a bed) & provide necessary management; such as Oxygen, suction, nebulizer, IV fluids, IV medications, transfusion, consultation, urgent laboratory &/or imaging studies Some will need admission for deterioration (?ICU) or prolonged stay
D	Needs urgent services diagnostic tests, treatment(s) &/or observation	Perform necessary management in ER; such as Observation, consultation, urgent laboratory &/or imaging studies, nebulizer, IM or IV medications, transfusion, ORT Most can stay in waiting area, but provide for • Easy accessibility • Reassessment
E	To be managed as outpatient	Send home with proper plan

CPR - ADVANCED LIFE SUPPORT

INDICATION: Cardiopulmonary arrest or near arrest (pre-arrest).

CPR should be performed immediately on any child who has become unconscious, is not showing any signs of life and is found to be pulseless. Signs of life refer to spontaneous movement, response to stimuli (including cough/gag on attempting airway-breathing support) and normal spontaneous breathing (NOT gasping)

STEPS: ABCDEF

Basic life support (ABC) can be provided anywhere without specialized equipment.
ABC support can also be performed using equipment and an advanced airway eg endotracheal tube.
Also, DEF are components of advanced life support.
Correctable factors should be addressed

A: Airway: Open - Remove FB if any-clear and maintain the airway. Insert endotracheal tube if/ when equipment and experience available.

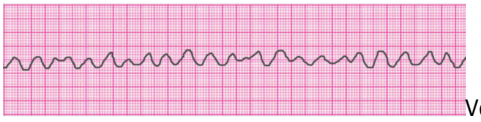
B: Breathing: Maintain by assisted ventilation (mouth to mouth, bag and mask, subsequently bag and endotracheal tube). Use oxygen if/ when available.
Most cases of cardiac arrest in children are secondary to a respiratory compromise. An unconscious child may therefore require respiratory (airway + breathing) support if he has apnea/ irregular gasping with a still beating heart.

C: Chest compressions should be started immediately if there is no central (eg Carotid) pulse, followed by respiratory (AB) support.

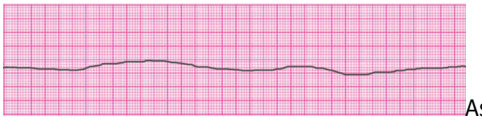
D: Drugs mainly adrenaline. IV is preferable. ET route is used if no IV access (less effective)

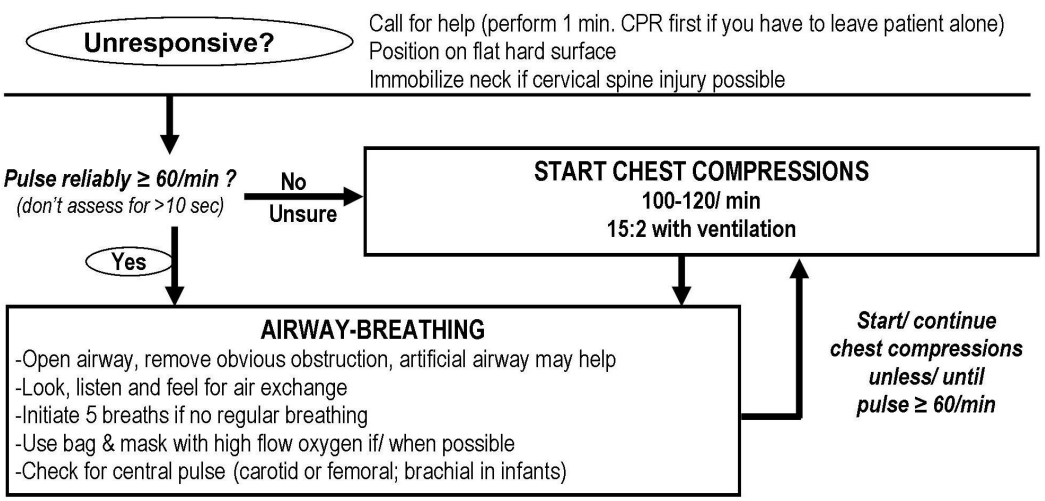
E: ECG monitor to determine cardiac electrical activity:
-asystole, pulseless electrical activity (electromechanical dissociation), extreme bradycardia
-shockable rhythms (managed by DC shock/ defibrillation): ventricular fibrillation and ventricular tachycardia

F: Defibrillation for shockable rhythms. *Provide 4J/Kg shock and minimize interruptions to CPR. After 3 shocks, give low dose adrenaline and amiodarone*

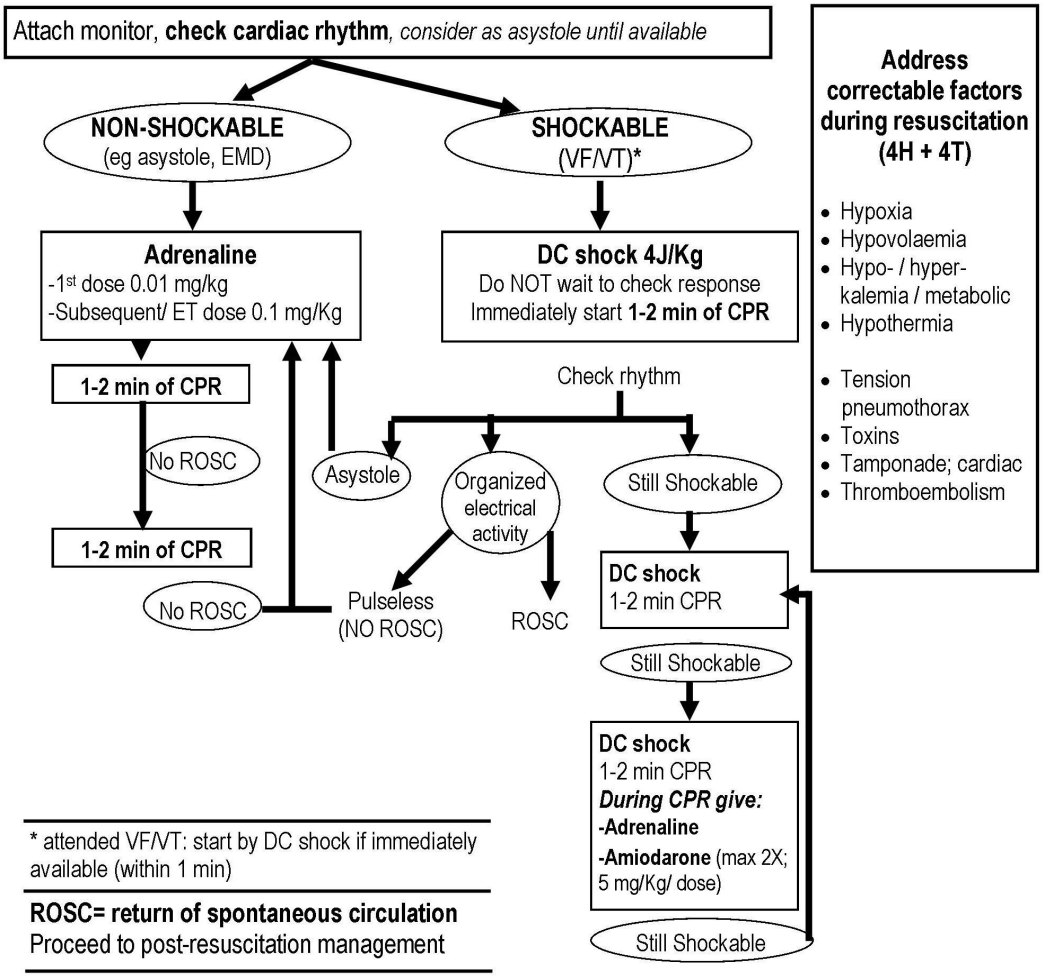

Ventricular fibrillation


Ventricular tachycardia


Asystole



Provide continuous CPR Intubate trachea if/ when possible without excessively interrupting CPR.
*Once intubated, provide **continuous** chest compressions; breaths do not have to alternate with compressions then (breath rate 12/min, increase to 20 after ROSC, higher in neonates eg 20 increase to 30 after ROSC)*



* attended VF/VT: start by DC shock if immediately available (within 1 min)

ROSC= return of spontaneous circulation
Proceed to post-resuscitation management

EMD= electromechanical dissociation,, VF= ventricular fibrillation, VT= ventricular tachycardia

Emergency equipment sizing by age

Endotracheal intubation:

Age	Weight (Kg)	Blade	ETT	***Depth of ETT*** **Newborn: 6 + weight (kg)** **Others: 3 X size of ETT**
Neonate		0-1	3	
Infant	5	1	3.5	
Toddler	10	1-2 or 1.5 Wis-Hipple	4.5	
6 years	20	2	5	***Size of ET tube*** **Uncuffed tube: (Age/4) + 4** **Cuffed tube: (Age/4)**
9 years	30	2	6	
Adolescent	50	3	7	
Adult	>50	3	8	

EQUIPMENT SIZING CHART

Equipment	< 3Kg	3-5 Kg	Small Infant 6-7 Kg	Infant 8-9 Kg	Toddler 10-11 Kg	Small child 12-14 Kg	Child 15-18 Kg	Child 19-23 Kg	Large Child 24-29 Kg	Adult or ≥30Kg
Resuscitation Bag	Infant	Infant	Infant/ Child	Infant/ Child	Child	Child	Child	Child	Child	Adult
Oxygen Mask (NRB)			Pediatric	Pediatric	Pediatric	Pediatric	Pediatric	Pediatric	Pediatric	Pediatric/Adult
Oral Airway (mm)			50	50	60	60	60	70	80	80
Laryngoscope Blade (size)	0-1 Straight	1 Straight	1 Straight	1 Straight	1 Straight	2 Straight	2 Straight	2 Straight or Curved	2 Straight or Curved	3 Straight or Curved
ET Tube (mm)	< 1 Kg 2.5 1-2 Kg 3.0	3.5	3.5 3.0 Cuffed	3.5 3.0 Cuffed	4.0 3.5 Cuffed	4.5 4.0 Cuffed	5.0 4.5 Cuffed	5.5 5.0 Cuffed	6.0 Cuffed	6.5 Cuffed
ET Tube insertion Length (cm)	0.5 Kg: 6.5 1 Kg: 7 2 Kg: 8	3 Kg: 9-9.5 4 Kg: 9.5-10 5 Kg: 10-10.5	10.5-11	10.5-11	11-12	13.5	14-15	16.5	17-18	18.5-19.5

Equipment	< 3Kg	3-5 Kg	Small Infant 6-7 Kg	Infant 8-9 Kg	Toddler 10-11 Kg	Small child 12-14 Kg	Child 15-18 Kg	Child 19-23 Kg	Large Child 24-29 Kg	Adult or ≥30Kg
Suction Catheter (F)	6	6-8	8	8	10	10	10	10	10	10-12
BP Cuff	Neonatal	Neonatal #5/ Infant	Infant/ Child	Infant/ Child	Child	Child	Child	Child	Child	Small Adult
IV Catheter (ga)	24	22-24	22-24	22-24	20-24	18-22	18-22	18-20	18-20	16-20
IO (ga)			18/15	18/15	15	15	15	15	15	15
NG Tube (F)			5-8	5-8	8-10	10	10	12-14	14-18	16-18
Urinary catheter (F)		5	8	8	8-10	10	10-12	10-12	12	12
Chest Tube (F)		8-12	10-12	10-12	16-20	20-24	20-24	24-32	28-32	32-38

Abbreviations: BP, blood pressure; ET, endotracheal; F, French; IO, intraosseous; IV, intravenous; NG, nasogastric; NRB, nonrebreathing; ga, gauge; mm, millimeters; cm, centimeters

SYSTEM FAILURE AND SYSTEM SUPPORT

System	Assessment	Failure	Support
Resp-iratory	**-Respiratory distress** **-Respiratory weakness** **-Ventilation (pCO_2)** **-Oxygenation** **-Pulmonary gas exchange** (PO_2/FiO_2 ratio, O.I.) **-Chest findings**	**-Type I: lung failure/ hypoxic respiratory failure** **-Type II: pump failure/ hypercapneic respiratory failure/ acute respiratory acidosis** Due to respiratory depression, weakness or exhaustion	**-Upper airway** **-Oxygen therapy** **-Mechanical ventilation** **-ECMO** + therapy for bronchospasm/infl. + chest care + FB removal +drainage of pleural collections (pneumothorax, large effusions)
Cardio-vascular	**-Tissue perfusion** **-BP** **-Vascular tone** **-Cardiac performance** (rhythm, contractility, SV/CO) **-Circulatory filling**	**-Shock (acute circulatory failure):** Tachycardia, poor peripheral perfusion, hypotension **Others:** **-Acute heart failure** Tachycardia, tachypnea and tender liver; systemic & pulmonary congestion **-Hypertensive emergency/ urgency** **-Arrhythmias**	**-Oxygen ± M.V.** **-Volume** **-Medications (vasopressors, inotropes, VD)** **-Mechanical (balloon, VA-ECMO)** + management of arrhythmias, hypertension, HF + drainage (tamponade & tension pneumothorax)
Neuro-logic	**-Level of consciousness** **-Seizures** **-Increased ICT** **-Specific/ focal neurological disorders**	**-Coma:** unconsciousness and not arousable by painful stimuli **-Status epilepticus**	**- ABC** **- Control convulsions** **- Control ICT** **- Correct metabolic abnormalities.** Consider IEMs, infectious & toxic causes **- Care of the comatose**
Meta-bolic	**-Hypothermia & hyperpyrexia** **-Hypoglycemia** **-Fluid, electrolyte & acid-base disturbances** **-Diabetic ketoacidosis, acute adrenal insufficiency** **-Acute kidney injury, acute hepatic failure**		**- Correction of metabolic disturbances** **- Replacement therapies eg dialysis**
Hema-tological	**-Hb/ RBCs** **- Thrombocytopenia** **-Leucopenia** **- Thrombophilia** **-Bleeding** **- Thromboembolism** **-Coagulopathy**		**- Control of bleeding** **- Transfusions** **- Anticoagulation/ thrombolytic therapy** **- Stimulating agents** eg G-CSF
Infection	**-Clinical** **-Laboratory** **-Microbiological**		**Prevention & treatment of infections**
Nutrition	**-Clinical** **-Anthropometric** **-Laboratory –Intake**		**Nutritional support**
Others			**Ambulation, physiotherapy, psychiatric support, etc.**

Infection, nutrition, etc are not system failures in strict sense of the term; however, are required supports.

INFECTION:

- Both a cause & a complication of critical illness
- Infection control precautions should be followed at all times
- Unnecessary devices should be removed & non-critical patients should be discharged
- Monitor patients for infections & obtain necessary cultures, before starting or modifying antibiotics & during fever if appropriate
- Use common sense, a plan & culture results when prescribing antibiotics
- Consider interactions when prescribing multiple antibiotics
- Reevaluate antibiotic prescriptions after 2-5 days based on response & microbiology results. STOP UNNECESSARY antibiotics and plan the required DURATION of therapy

NUTRITION:

- Enteral feeding, as early as possible after stabilization, mostly within 24 hrs, is the aim
- Neither MV, stable inotropic support nor coma are contraindications to enteral feeding
- Escalate as tolerated, minimize unnecessary interruptions
- For fluid calculations, 85% of your formula is fluid
- Parenteral when enteral not possible, not mutually exclusive

MANAGEMENT CYCLE

- **Assessment → Plan**
- **Intervention**
- **Monitoring/ Reassessment**
- **Continue, modify or withdraw intervention**

- **CONSIDER TRENDS/ NOT JUST SNAPSHOTS (unless they are extreme)**
- **CONSIDER LEVEL OF SUPPORT when judging patient's condition/ progress**
- **TIMELY INTERVENTION is very important. Do it when it would benefit. Not interfering is a decision.**
- **We do not LIKE to be invasive, although we sometimes HAVE TO**
- **Consider physiology and do NOT UNNECESSARILY SUPPRESS IT (eg MV, nutrition,etc)**
- **Make the ICU environment LESS HOSTILE: consider the environment, promote safety, involve parents appropriately, control pain, rehabilitate early, etc**

RESPIRATORY DISTRESS, FAILURE & MONITORING

RESPIRATORY DISTRESS

Definition: **Increased respiratory effort manifesting as difficulty in breathing**

Causes:

Pulmonary/ airway		**Non-pulmonary causes**
Pneumonia	Aspiration syndromes	Heart failure
Acute asthma attack	Pneumothorax	Metabolic acidosis
Bronchiolitis	Obstructive emphysema	(all causes …)
Pulmonary edema	Lung collapse	Acute severe anemia
ARDS	Pleural effusion	Central hyperventilation
Neonatal hyaline membrane disease	Bronchopulmonary dysplasia	
	Bronchiolitis obliterans	
	Organophosphorous poisoning	

Clinical grading (clinical picture):

Grade I: tachypnea & working alae nasi
Tachypnea: RR above 60/ min. in neonates & infants < 2 Mo, 50/ min. in infants 2-12Mo, 40/ min. in children 1-5 yrs, 30/ min. in older children & 20/ min. in adolescents
Grade II: intercostal & subcostal retractions
Grade III: grunting
Grade IV: respiratory failure (clinically cyanosis & disturbed consciousness)

-Grades I-III represent progressive compensatory effort to prevent respiratory failure
-Severe RD may be denoted by MARKED tachypnea, SEVERE retractions or GRUNTING
-Respiratory failure occurs with severe hypoxia or exhaustion
-Because cyanosis and disturbed consciousness are late signs of hypoxia, patients may already have respiratory failure (blood gases) before grade IV (clinical)

Management:

-Assess and monitor respiratory effort, oxygen saturation and blood gases for development of respiratory failure
-Respiratory support
-Treatment of the cause
-Do not forget non-pulmonary causes!

RESPIRATORY FAILURE

Definition: **Inability of the respiratory system to maintain oxygenation or ventilation (CO_2 elimination)**

More than 50% of deaths in the PICU are respiratory in origin.

Types of Respiratory failure:

	Type I: Lung failure Oxygenation failure Hypoxic RF	**Type II: Pump failure Ventilation failure Hypercapnic RF**
Basic defect	Oxygenation (severe hypoxemia)	Hypoventilation (hypercapnia)
Causes	➢ **<u>Airway/ lung disease</u>** (causes of RD)	1) Respiratory depression: CNS infection, intracranial hemorrhage, drugs/ toxins 2) Respiratory muscle paralysis: ▫ Guillain Barre Syndrome. ▫ Poliomyelitis ▫ Myasthenia ▫ Werding Hoffman disease 3) Respiratory muscle fatigue: Severe type I RF
Clinical	-Respiratory distress (…) -Chest signs of the cause	-Shallow irregular breathing or apnea -The cause (neurological or respiratory)
ABG	Severe hypoxemia (low PaO_2) Hypoxic <u>metabolic</u> acidosis <u>±</u> hypoventilation	Hypoventilation (high $PaCO_2$) Acute respiratory acidosis <u>±</u> hypoxemia

Management:

- Respiratory monitoring
- Respiratory support:
 - Oxygen therapy
 - Positive pressure support (ventilation)
 - Suction, aerosol therapy
- Specific treatment of the causative disease

RESPIRATORY MONITORING

A. Clinical Monitoring:

- Respiratory rate (RR) & heart rate (HR)
- Degree of RD
- Color of the patient
- Peripheral perfusion
- Chest signs

B. Oxygen Saturation (SaO2): using Pulse Oximeter

- **Advantages:** Simple, reliable & non-invasive
- **Disadvantages:**
 - -Cannot measure hyper oxygenation
 - -False reading in Shock –very dark skin –Methemoglobinemia
- **Interpretation:**
 - >95%: Normal
 - 90-95%: Mild hypoxemia
 - 85-90%: Moderate hypoxemia
 - <85%: Severe hypoxemia

C. Arterial Blood Gases:

- **Advantages**
 - Assessment of oxygenation (PaO_2)
 - Assessment of ventilation (CO_2 elimination)
 - Assessment of acid-base status
- **Disadvantages:** Invasive and painful, expensive
- **Normal values**

PaO_2	90-100 mm Hg
$PaCO_2$	35-45 mm Hg
pH	7.38-7.42
HCO_3	22-26 mEq / L (infants 20-24)

- **General principles:**

-Arterial BG can assess acid base status, ventilation & oxygenation

-Venous BG can assess acid base status, ventilation (pCO_2 above arterial by 6mmHg if perfusion is normal) but not oxygenation

-Mixed venous or central venous SATURATION is a surrogate for tissue perfusion IF ARTERIAL OXYGEN CONTENT (Hb and pO_2) IS NORMAL

-Arterialized capillary samples can assess acid base status, ventilation and oxygenation (with different normal pO_2 values)

ABG should be interpreted in relation to the clinical condition & level of support:

- Normal ventilation can be achieved with severe distress (compensation)
- Normal (or hyper-) oxygenation can be achieved with O_2 therapy

Interpretation of arterial blood gases

Ventilation	***Check PaCO2***	**<35 mmHg ➔ hyperventilation** **35-45 mmHg ➔ normal** **>45 mmHg ➔ hypoventilation** *>45-50= mild, >50-60= moderate, >60= severe*
Oxygenation	***Check PaO2***	**>100 mmHg (hyperoxygenation)** *may occur with O2 therapy, hyperventilation or air in sample* **90-100 mmHg ➔ normal** **<70 mmHg ➔ hypoxemia** **<55 mmHg ➔ severe hypoxemia (RF type I)**
Acid base status	***pH, PaCO2 & bicarbonate*** *must be checked to judge acid-base*	**Low pH ➔ acidosis** **High pH ➔ alkalosis**
	Acidosis	**High CO2 ➔ respiratory acidosis** *+ Normal bicarbonate: acute (uncompensated)* *+ Increased bicarbonate: chronic* *(renal compensation by base retention in attempt to get pH near normal, takes 24hrs)*
		Low bicarbonate ➔ metabolic acidosis *+ CO2 should be low* *(rapid respiratory compensation by hyperventilation, takes minutes)* ***Predicted CO2 = bicarbonate X 1.5 + 8***
		Low bicarbonate with high CO2 and very low pH ➔ mixed metabolic & respiratory acidosis ***Also, a low pH with near normal CO2 & bicarbonate denotes mixed acidosis***
	Alkalosis	**+ high bicarbonate ➔ metabolic,** **+ low CO2 ➔ respiratory**

Pulmonary gas exchange (ventilation-perfusion mismatch) can be assessed by:

- **Oxygenation index (in patients on MV only):** FiO2 X MAP / PaO2
 (MAP= mean airway pressure)
 <5 is a requirement for extubation & >20-25 denotes severe lung disease
- **Arterial/ inspired oxygen ratio:** PaO2/ FiO2 (P/F ratio)
 (FiO2 as a fraction i.e. 100% =1, room air =0.21)
 <300 denotes impaired gas exchange & <100 denotes severe lung disease

ANION GAP Na – (Cl + HCO3) denotes extra unmeasured anions.

In patients with metabolic acidosis:

- Normal anion gap ➔ bicarbonate loss eg GE, RTA
- High anion gap (>12) ➔ addition of fixed acid (uremia, DKA, organic acidurias, lactate, etc)

INTRODUCTION TO MECHANICAL VENTILATION

THIS MACHINE HAS NO BRAIN; USE YOURS!

MV is the INTERACTION between the patient and ventilator to achieve the objectives. You cannot be focusing on the ventilator and neglecting the patient. Patients change and so should the way of support. MV is supposed to be temporary; treat the underlying cause and aim to get your patient off as soon as possible, the proper time to consider this is AS SOON AS YOU VENTILATE THE PATIENT.

OBJECTIVES:

To achieve appropriate

- Ventilation
- Oxygenation
- Work of breathing
- Lung protection

Ventilation is the gas exchange between alveolar and surrounding gas. Minute volume is the volume exchange per minute, tidal volume is per breath.

Minute volume = tidal volume x respiratory rate

High pCO_2 denotes inadequate minute volume (hypoventilation)

Note that part of the volume delivered is to dead space so minute volume increases more by increasing an inadequate tidal volume increases than by increasing rate

Excessive tidal volume is the main cause of ventilator associated lung injury

Oxygenation at the level of arterial pO2 depends on ventilation and other factors:

- Inspired pO2: barometric pressure and FiO_2 (= % oxygen in inhaled gas mixture)
- Mean airway pressure
- Pulmonary gas exchange; affected by:
 - *Effective gas exchange area (amount of functioning= recruited lung)*
 - *Ventilation-perfusion match (=perfused non-ventilated alveoli)*
 - *Diffusion*

Note that at the level of tissues it also depends on hemoglobin, cardiac output, blood flow and tissue oxygen utilization

Excessive or non-synchronized (not in harmony with ventilator) **work of breathing** is a problem; however, totally eliminating patient efforts is usually NOT the goal.

The patient is receiving the TOTAL of his effort and the ventilator effort. The total should provide the required minute volume. The relation between total effort and minute volume depends on airway resistance (needs more effort) and lung compliance (higher compliance needs less effort)

MV can cause lung injury in different ways:

- Volutrauma (=overdistension)
- Barotrauma (=high ΔP; i.e. difference between PIP and PEEP)
- Atelectrauma (=repeated collapse and reopening of alveoli each breath)
- Oxygen toxicity (depends on FiO_2 and time)
- Infection (eg VAP= ventilator associated pneumonia)

Others as aspiration, hypoxic events, etc

INDICATIONS *Consider the trend and anticipate progress*

- Apnea, obviously ineffective breathing (absolute)
- Controlled GA with NMB (absolute, most common)
- Severe hypoxemia despite oxygen therapy (absolute)
- Severe hypoventilation (pH & pCO2) (absolute if acidemic with pCO_2 >60)
- Markedly ↑ ICP (strong)
- Refractory shock (strong)
- Deep coma, refractory status epilepticus (relative if no other indication)
- Unacceptable or deteriorating work of breathing (relative but most common in ICU)

WHAT DOES THE VENTILATOR DO?

- Blends air and O_2 (FiO_2) *GAS MIXTURE MUST BE HUMIDIFIED AND WARMED*
- Provides CPAP/ PEEP
- Gives assisted/ controlled breaths
- Detects patient's effort (to trigger assisted breaths)
- Sensors, monitors and alarms

PEEP/ CPAP:

Positive pressure provided at the end of expiration (i.e. does NOT reach zero), with the aim of maintaining alveoli opened and preventing end-expiratory collapse, leading to increased lung recruitment and better oxygenation.

CPAP= the same applied to SPONTANEOUSLY breathing patient; would also reduce respiratory distress.

It can increase air trapping especially with bronchial obstruction and can increase intracranial pressure and reduce venous return.

RESPIRATORY CYCLES:

Cycle = breath = inspiration and expiration

Expiration is generally passive; Cycling = end of inspiration and beginning of expiration

Types of cycles

SPONTANEOUS *PATIENT ONLY* Patient determines onset, does all effort and determines duration of the breath Ventilator has no role (except possibly PEEP and FiO_2)	**ASSISTED** *SHARED EFFORT* Ventilator does part or all the effort, while patient determines the onset and possibly the duration of the breath	**CONTROLLED/ MANDATORY** *VENTILATOR ONLY* Ventilator determines onset, duration and does all the effort

Cycle phases

A. INITIATION ***When breath starts*** - Time (60/ rate) - Trigger (patient effort) by pressure or flow - Combined (in SIMV mode)	**B. CONTROL/ LIMIT** ***How much effort?*** - Pressure (PIP) - Volume (tidal vol) The other is the result - Combined (in Dual modes)	**C. CYCLING** ***End of inspiration*** - Time (insp. Time) - Flow (patient termination, in PSV mode)

BASIC MODES

CMV *Continuous (controlled) mandatory ventilation*	All cycles controlled at set rate, all effort by ventilator, NO patient contribution *May be pressure or volume controlled*
SIMV *Synchronized intermittent mandatory ventilation*	The set rate is given - As assisted cycles at the time initiated (triggered) by patient; or - As controlled cycles if patient did not trigger Patients' efforts EXCEEDING the set rate remain spontaneous *May be pressure or volume controlled*
AC/ PTV/ SIPPV *Assist-control*	ALL PATIENT EFFORTS trigger assisted cycles (ventilator rate = patient rate) Set rate is a BACKUP; will be given as controlled cycles if patient did not trigger *May be pressure or volume controlled*
PSV *Pressure Support*	Patient efforts trigger assisted cycles (as AC); but Inspiratory time is determined by the patient *Always pressure; may be alone or with SIMV*

SETTINGS

Mode	Synchronized modes (eg AC or SIMV/PSV) better if patient has adequate drive. *CMV when patient CANNOT, SHOULD NOT or WILL NOT contribute*
PEEP	Best PEEP is that which provides best oxygenation without adverse effects *Classically 5, may increase for stiff lungs, minimize for obstructive pathology and ICT*
FiO2	As needed for adequate oxygenation *Start high (80-100%) in hypoxic or shocked patients, Normal lungs require lower FiO2 (40%)* Reduce as tolerated, Transiently increase for desaturation while correcting cause
Inspiratory time	Inspiration: expiration (I/E) ratio is 1:2; Insp. Time approx. 20/ rate *Use PHYSIOLOGICAL or ACTUAL TOTAL (vent + pt) rate; whichever is GREATER* May use I/E 1:1 for stiff lungs but care for incomplete expiration in obstructive pathology
PIP	*In P controlled/ limited modes:* PIP is the MINIMUM that achieves adequate TIDAL VOLUME (measured or assessed by air entry and chest expansion) *It is the PIP-PEEP difference (*Δ P*) that matters and this is what you set is PSV*
Tidal volume	*In V controlled/ limited modes:* set at 6-8 mL/ Kg, neonates may require a bit higher, consider ETT leak and circuit compression
Flow	If required, set the flow that will achieve desired PIP *in proper time* / desired tidal volume *during inspiratory time* (2-3 L/ min in infants and 1-2 L/ min in older patients)
Trigger sensitivity	*The –ve pressure or flow required for the ventilator to recognize patient effort* Less –ve P, less flow = more sensitive= recognize LESS effort= higher ventilator rate *Set the trigger that will recognize MOST patient efforts and will not cause extreme tachypnea or false trigger*

Mean airway pressure (MAP) is the average P during the cycle (insp and exp.). It depends on PEEP, PIP and I/E ratio

VENTILATING STIFF LUNGS	VENTILATING OBSTRUCTIVE CASES
Low compliance; massive pneumonia, ARDS, etc ● Adequate PEEP & MAP for recruitment ● Guided by oxygenation & P/V loops ● Minimal Δ P needed to achieve acceptable tidal vol. (and it will be high !!!!) ● Adequate Ti (needed to maintain adequate T.V. & oxygenation without excessive Δ P) ● Exp. time usually not a problem (short time constant)	*Bronchospasm, bronchiolitis, etc* ● Treat the cause !!!!! ● Beware the alveoli are already distended (autoPEEP?) ● Avoid excessive PEEP ● ADEQUATE exp. time is the clue ● This may limit rate &/or delivered tidal volume so permissive hypercapnea may be needed esp with also stiff lungs

ASSESSMENT & ADJUSTMENT

MONITORING

- Ventilation
- Oxygenation
- Work of breathing (Pt+Vent roles)
- Readiness for weaning

ADJUSTMENT

Inadequate ventilation	Inadequate oxygenation	↑ or ↓ patient contribution
● Ensure adequate Tidal volume; then ● Increase rate *Care of I/E ratio*	● Ensure adequate ventilation ● Increase FiO_2 ● Recruitment ● MAP (mainly PEEP, also MAP by I/E ratio or PIP) *Care of excessive O_2/ pressures*	● Question sometimes difficult ● If you can lower support and the patient can make up the difference ● Commonly trial and adjustment

ACUTE DETERIORATION

Classically DOPE

- Displacement
- Obstruction
- Pneumothorax
- Equipment failure

There are others (some less acute) *eg asynchrony, derecruitment, loss of pt role after sedation/ NMB, bronchospasm, original pathology, VAP, …*

Deterioration on M.V.

PATIENT	TECHNIQUE
• Asynchrony • Barotrauma • Bronchospasm • Collapse • VAP	• Inlet pressure • Machine failure • Settings • Circuit block/ leak • Accidental extubation • Obstructed tube

MANAGING THE PATIENT

- Circuit and tube care
- Sedation
- Monitoring
- Infection control
- Management of causative disease
- Management of other systems/ conditions
- Management of problems
- Weaning and extubation
- Post-extubation care & monitoring

WEANING and EXTUBATION

- Is the cause of respiratory failure gone or getting better ?
- Is the patient well oxygenated and ventilated ?
- Can the heart tolerate the increased work of breathing ?
- Are there correctable factors that can cause failure?

- **Extubation readiness**

There is no guarantee that a patient will extubate successfully even when following these suggestions. Always be prepared to re-intubate if needed.

- Ventilator settings: minimal PEEP (5), minimal oxygen requirement, low rate (5-10) unless short term ventilation, tolerating low pressure support (0-10) trial
- Adequaate consciousness with minimal sedation requirements & no/ acceptable pain
- Control of airway reflexes
- Patent upper airway (air leak around tube?). Consider steroids (unless short intubation)
- Review cardiopulmonary status

Immediately prior to extubation:

- Consider preoxygenation (higher FiO2) unless limiting oxygen
- Suction ETT prior to extubation and have supplies ready to suction upper airway immediately after extubation
- Oxygen/CPAP/BiPAP - have supplies ready in the room if you anticipate a transition to anything but room air
- Head of bed elevated
- Aerosols - have racemic epinephrine ready. Consider salbutamol if patient had tight chest

SHOCK AND HEMODYNAMIC SUPPORT

Shock: failure of the circulatory system to maintain adequate tissue perfusion
➔ inadequate delivery of oxygen and nutrients to meet tissue metabolic demands

Adequate perfusion depends on:
Blood volume ➔ Cardiac filling ➔ Myocardial contractility ➔ SV (&CO) ➔ Vascular tone ➔ BP
NB In addition to perfusion, oxygen content (Hb & O2 saturation) is essential for tissue oxygenation

<table>
<tr><th>Causes of shock</th><th>Grades/ stages</th></tr>
<tr><td>HYPOVOLEMIC
bleeding, severe dehydration, burns, capillary leak</td><td rowspan="2">EARLY= hypoperfusion
- Slow capillary refill (>2sec)
- Cold extremities (core-peripheral temp >3)
- Mottling, peripheral cyanosis
- ↓ urine output, ↑ lactate
- Tachycardia & tachypnea (compensatory)</td></tr>
<tr><td>DISTRIBUTIVE
Neurogenic, anaphylactic & warm septic shock</td></tr>
<tr><td>CARDIOGENIC
Poor contractility with myocardial disease or ischemia, sepsis & MOSF</td><td>ESTABLISHED= hypotension
(systolic hypotension denotes DECOMPENSATION)</td></tr>
<tr><td rowspan="2">OBSTRUCTIVE
-Cardiac tamponade & tension pneumothorax
-Massive pulmonary embolism, critical stenotic lesions</td><td>ADVANCED
Severe &/ or prolonged shock with MOSF</td></tr>
<tr><td>IRREVERSIBLE</td></tr>
<tr><td colspan="2">NB SEPTIC SHOCK is due to systemic inflammatory response with loss of vascular tone, myocardial depression & capillary leak (to different degrees in different patients)
Shock can have MIXED &/or CHANGING pathophysiology</td></tr>
</table>

MANAGEMENT OUTLINE FOR SHOCK

I. AIRWAY-BREATHING support Ensure/ secure airway, Oxygen therapy
MV for refractory shock, resp. exhaustion, unacceptable resp. effort or usual indications

2. EMERGENCY VASCULAR ACCESS & CORRECT urgent issues
- VA ideally secured before the development of shock (anticipate)
- Attempt central, femoral or IO if no access for few min.
- A small peripheral line is better than none
- A CVC will be needed for monitoring & vasopressors in refractory cases

Identify & correct abnormalities in temperature, glucose, Ca, arrhythmias, cardiac tamponade, tension pneumothorax, initiate broad-spectrum antibiotics for suspected sepsis, adrenal insufficiency (catecholamine-resistant cases should receive 50-100mg/m2 hydrocortisone), maintain Hb ≥10g/dL and albumin ≥3g/dL in all volume-refractory cases, urgent surgery may be needed to control blood loss

3. VOLUME EXPANSION
20 mL/ Kg normal saline rapidly
-Volume is given unless/ until patient has evidence of circulatory overfilling/ overload *(neck veins, development of rales or hepatomegaly, CVP>8-12, non-collapsible IVC, etc)*
-Smaller boluses (5mL/Kg) in cases of cardiogenic shock or very poor contractility (fluid-sensitive)
-Repeat as needed. Consider pharmacological therapy after 1-2 boluses (40mL/Kg) or earlier if there is poor contractility or overfilling/ overload
-Use blood/PRBCs or plasma only if otherwise indicated. Use 5% albumin for hypoalbuminemic patients & for those with capillary leak and poor response to the first fluid bolus

Poor response to volume

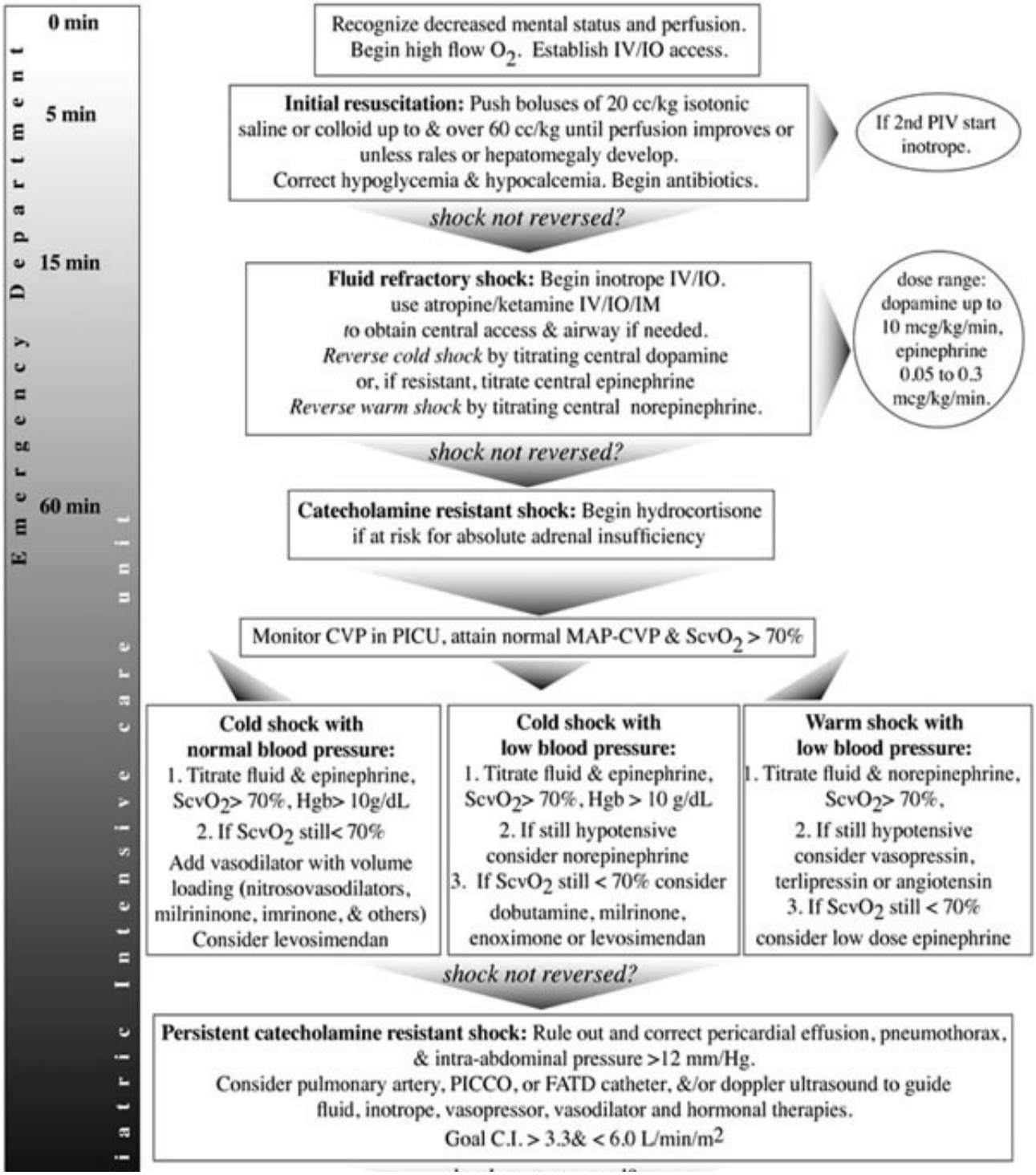
↓vascular tone *(neurogenic, warm septic)*	**Persistent hypovolemia** (capillary leak, ongoing loss) ➔Volume, ?Vasopressor	**↓ CO despite adequate filling** (Exclude obstructive shock)
↓SVR, ↑pulse pressure, ↑ or normal CI, ScvO2>70%		**MYOCARDIAL DYSFUNCTION** ↑CVP, ↓SV/CI, ↓FS, ScvO2 <70%

4. PHARMACOLOGICAL THERAPY

VASOPRESSORS	**INOTROPES**
-Dopamine 10ug/Kg/min -Noradrenaline -Vasopressin/ terlipressin *Dopamine use is currently declining*	-Dopamine 10ug/Kg/min -Adrenaline ***NB. VASODILATOR*** therapy to ↓ afterload is beneficial in those with **poor perfusion with ↑SVR and adequate volume** *Can cause hypotension; hence* ***Inodilators (***dobutamine, milrinone & levosimendan) may be used in cardiogenic shock & with catecholamines in septic shock ***Nitrates*** (no inotropic effect) may only be added to inotropes when BP is adequate

Check response & need for further volume, increasing doses &/or the use of different agents at max. 15 min. cycles

5. Consider VA-ECMO for refractory shock



"Surviving sepsis" algorithm for septic shock

Choices of inotropic/ vasoactive infusions

Condition	Agents to consider	Remarks
Distributive **eg warm septic shock**	- Volume - Vasopressors *Dopamine 10ug/Kg/min *Noradrenaline (may be preferable) *Vasopressin, Terlipressin	Relative hypovolemia & loss of vascular tone. There is compensatory ++ in CO
Cold septic shock	- Volume unless/ until overfilling - Inotropes (ino-presssors): *Dopamine 10ug/Kg/min *Adrenaline (may be preferable) * Add noradrenaline or vasopressin if hypotensive on max. inotropes - With adequate volume & BP, and persistently high SVR with poor perfusion: * Inodilator * Add nitrate	Predominantly low CO
Cardiogenic shock	- Optimize volume (small boluses with caution if needed) - Dobutamine - Milrinone - Levosimendan - If hypotension, adrenaline	Predominantly low CO, compensatory ++ in SVR. BP response to inodilators is variable.

Infusions for hemodynamic support

Agent	Dose	Mechanism of action	Contra-ctility	SVR	HR	Myocardial O2 demand
Adrenaline	0.01-1 ug/Kg/min	Inotrope, dose-dependent (>0.3ug/Kg/m) vasopressor (β & α agonist)	+++	+	+++	+++
Noradrenaline	0.01-1 ug/Kg/min	Vasopressor, minimal inotropic effect (mainly α agonist)	+	++	++	++
Dopamine	10 ug/Kg/min	Inotrope, dose-dependent vasopressor (β1 & α agonist)	++	+	++	++
Dobutamine	5-20 ug/Kg/min	Inotrope with some VD effect (β agonist)	++	↓	+	++
Milrinone	0.25-0.75 ug/Kg/min	Inotrope with marked VD effect (phosphodiesterase inhibitor)	++	↓↓		+
Levosimendan	0.05-0.2 ug/Kg/min x24h	Long-acting Inotrope with VD effect (Ca sensitizer)	+++	↓	+	↓
Vasopressin	0.05-5 mU/Kg/min	Vasopressor (V receptor agonist)		+++		

HYPERTENSIVE CRISIS

Severe acute hypertension that can cause rapid organ damage

- Hypertensive urgency= no acute target organ injury
- Hypertensive emergency= Acute target organ injury
 (hypertensive encephalopathy, hypertensive HF, pulmonary oedema, AKI)

Note:

- The target is lowering SBP by 25% in the first 8 h and normalization after 24 hrs

- Secondary hypertension is common in children, particularly those <6yrs old and those with severe hypertension, although primary hypertension is increasing nowadays

- Hypertensive crisis can complicate diagnosed hypertension or be the first presentation

MEDICATIONS FOR HYPERTENSIVE CRISIS

(many are used off-label for this indication in children)

- Hydralazine (direct VD) 0.2-0.6 mg/Kg IV, can be repeated every 4-12hrs

- Labetalol (α and β blocker) 0.2-1 mg/Kg IV bolus, 0.25-3 mg/Kg/hr

- Esmolol (short-acting β blocker) 100-500 mcg/kg/min

- Sodium nitroprusside 0.5-10 mcg/Kg/min

- Furosemide 1-2 mg/Kg IV (in HF or with others)

- Calcium channel blockers
 - -IV Nicardipine
 - -Nifedipine (titrate from 0.25 mg/Kg to avoid rapid unpredictable drop)

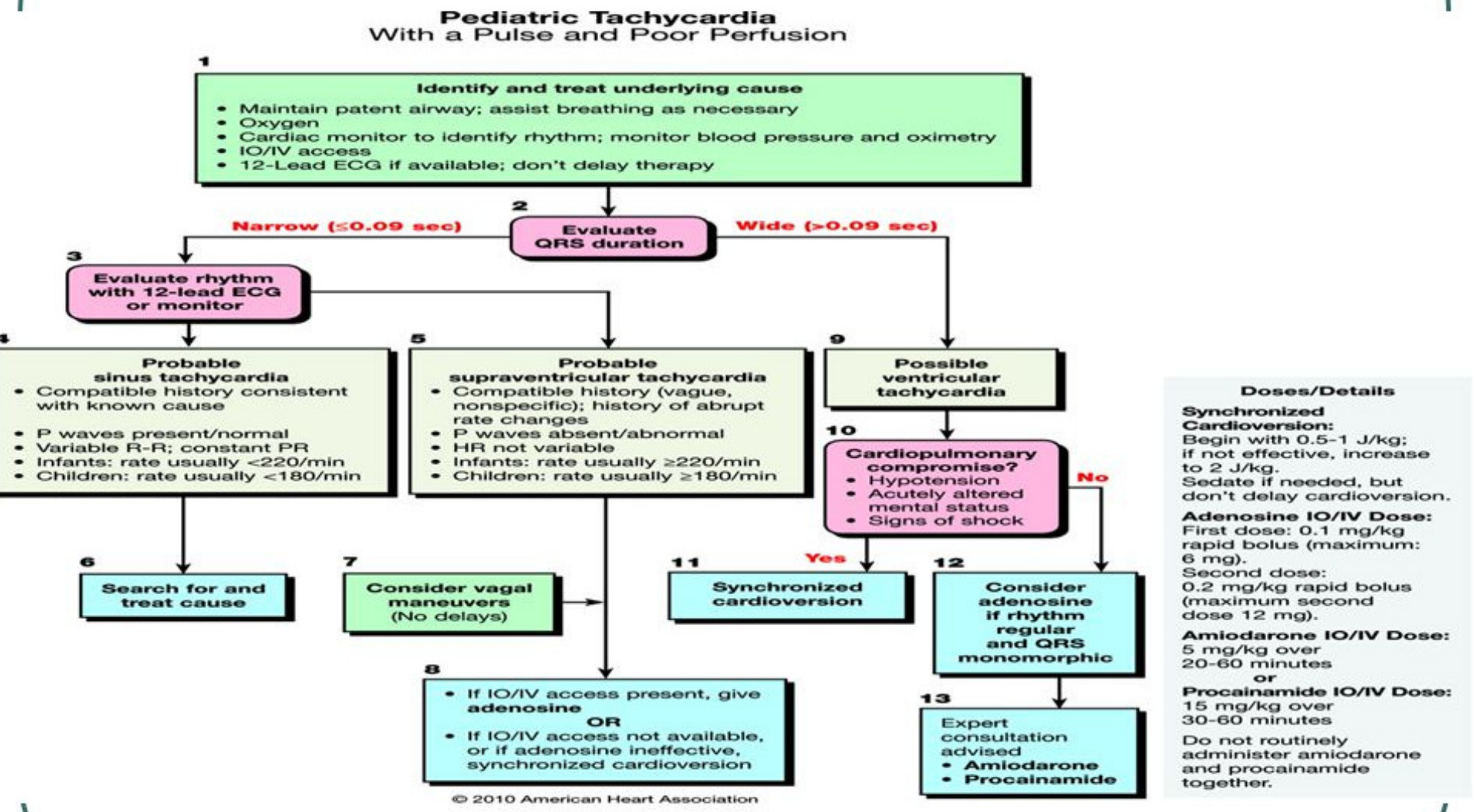
- Other oral agents as clonidine & ACE inhibitors

ARRHYTHMIAS: CONCEPTS

* Sinus rate upper limit is 220 in infants and 180 in children.
* In sinus tachycardia, treatment of the cause is the main line of therapy.
* Commonest type of pediatric tachyarrythmia is SVT (AV nodal reentrant tachycardia - AVNRT, AV reentrant tachycardia –AVRT- due to accessory pathway as in WPW syndrome)
* Adenosine is important in diagnosis of SVT (cases of confusing atrial tachycardia, atrial flutter and atrial fibrillation). It may terminate AVRT and AVNRT (only these 2 types of SVT respond to it)
* In any SVT or VT, assess the hemodynamic state of the patient
 1. **If stable, medical agents are helpful as amiodarone (SVT or VT) or lidocaine (VT only)**
 2. **If unstable, DC SYNCHRONIZED cardiovesion is done.**
* Pulseless VT and VF are managed according to advanced life support cardiac arrest protocol.
* The only situations which needs asynchronized DC (defibrillation) is VF
* In any arrythmia, check 3 items:
 1. Medications (inotropic agents)
 2. Mechanical factors (CVC, VA shunts)
 3. Electrolytes (Na, K, Ca, Mg and P)

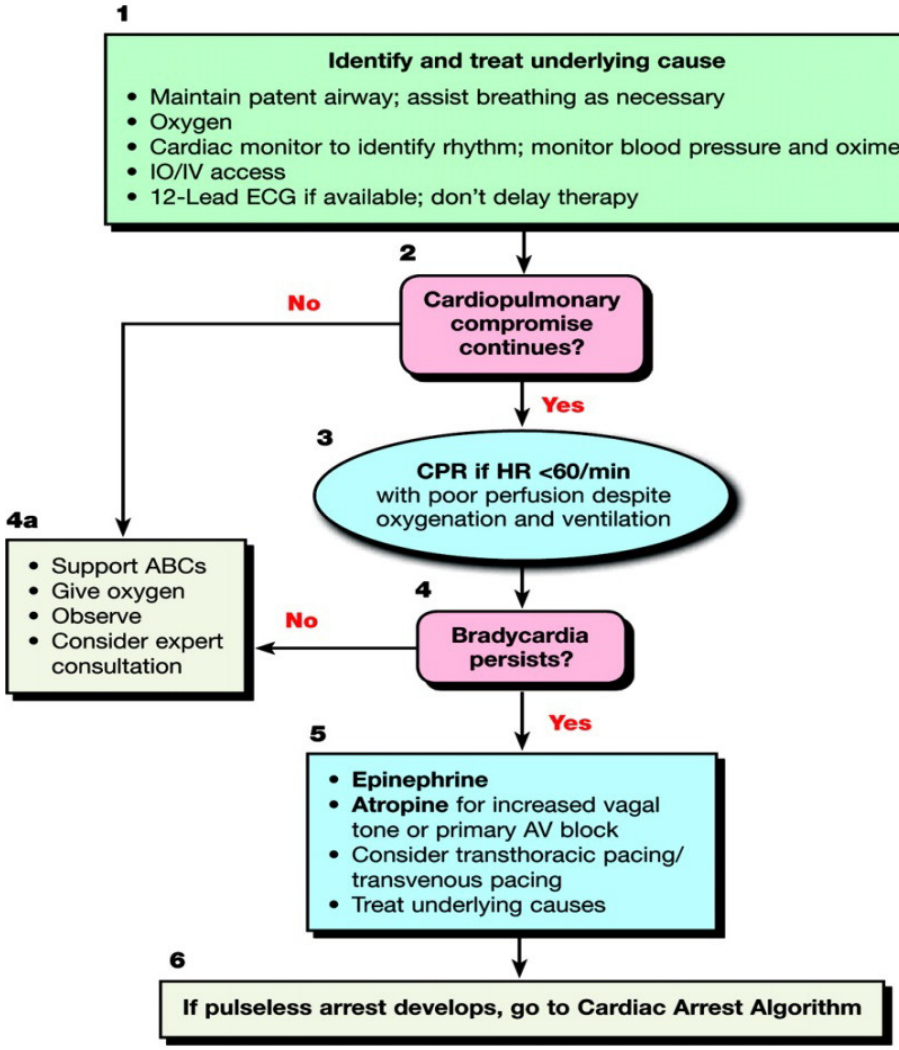
 With control of these factors, arrythmias can be controlled even without long term antiarrythmic drug administration.
* Any case of unexplained bradycardia, check the QT interval and causes of long QT (medications)
* If bradycardias are not associated with hemodynamic instability, don't give atropine, check electrolytes and medications first.
* The management of hemodynamically significant heart block is pacing

PALS Tachycardia Algorithm.



Kleinman M E et al. Pediatrics 2010;126:e1361-e1399

Pediatric Bradycardia
With a Pulse and Poor Perfusion



Cardiopulmonary Compromise

- Hypotension
- Acutely altered mental status
- Signs of shock

Doses/Details

Epinephrine IO/IV Dose:
0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If IO/IV access not available but endotracheal (ET) tube in place, may give ET dose: 0.1 mg/kg (0.1 mL/kg of 1:1000).

Atropine IO/IV Dose:
0.02 mg/kg. May repeat once. Minimum dose 0.1 mg and maximum single dose 0.5 mg.

COMA

CAUSES OF COMA

- Traumatic

- Accidental and non-accidental (child abuse)
- Concussion, contusion/ laceration, intracranial hemorrhage

- Hypoxic/ ischemic encephalopathy

- Perinatal asphyxia, Near drowning, Severe respiratory disease, Inhalation injury, Electrical injury, Severe/ prolonged shock, Prolonged CPR

- Intracranial infections Bacterial, viral, fungal, others (as leptospiral, spirochetal, trypanosome)

- Meningitis, Encephalitis, Brain abscess

- Metabolic

- Hypoglycemia
- Diabetic ketoacidosis
- Hyperglycemic hyperosmolar
- Electrolyte disturbances as Hypernatremia, Hyponatremia, Hypomagnesemia, Hypercalcemia
- Hepatic encephalopathy
- Uremic encephalopathy
- Acute adrenal insufficiency
- Hypothyroidism
- In-born errors of metabolism as Mitochondrial disorders, Urea cycle disorders, Organic acidurias

- Poisoning: Ethanol, narcotics, benzodiazepines, barbiturates, atropine, lead, etc.

- Vascular

- Hypertensive encephalopathy
- Cerebral vasculitis
- Stroke
- Intracranial hemorrhage

- Brain tumours

- Seizure-related: Post-ictal, Non-convulsive status epilepticus, Drug-induced

- Sepsis/ systemic infections

- Others

- Acute disseminated encephalomyelitis
- Myelinolysis
- Increased intracranial pressure
 (cerebral oedema, space-occupying lesions, acute hydrocephalus)
- Reye syndrome
- Psychogenic/ hysterical

Scores & grading are used to objectively assess consciousness and follow progress

Glasgow Coma Scale:

Adult			Pediatric	
Spontaneously	4	**Best Eye Opening**	Spontaneously	4
To verbal stimuli	3		To verbal stimuli	3
To painful stimuli	2		To painful stimuli	2
No eye opening	1		No eye opening	1
Oriented	5	**Best Verbal Response**	Appropriate coo & cry	5
Confused	4		Irritable cry	4
Inappropriate words	3		Inconsolable crying	3
Incomprehensible	2		Grunts	2
No verbal response	1		No verbal response	1
Obeys commands	6	**Best Motor Response**	Normal spontaneous	6
Localizes pain	5		Withdraws to touch	5
Withdraws to pain	4		Withdraws to pain	4
Flexion to pain	3		Flexion to pain	3
Extension to pain	2		Extension to pain	2
No motor response	1		No motor response	1

Murray score:

Best motor response as Glasgow	**(6 points)**
Brain stem responses	**(4 points)**
All intact	4
Some intact	3
Spontaneous respiration, no other	2
No spontaneous respiration	1

Grading of consciousness:

Normal	Awake, oriented, alert
Lethargic	Awake but not alert/ drowsy
Confused/ disoriented	
Coma grade I/ Stupor	Unconscious, briefly arousable to pain
Coma grade II	Unconscious, not arousable but responsive (reflex) to pain
Coma grade III	Unconscious, unresponsive to pain but spontaneous respiration present
Coma grade IV	Unconscious, unresponsive, no respiratory effort

Delirium = mental confusion + motor excitability

Initial evaluation of coma

-ABC's: assessment and stabilization

-Ensure adequate ventilation & oxygenation
-BP monitoring

-Draw labs: Glucose, lytes, ammonia, blood gas, LFTs, BUN/Cr, CBC, lactate, pyruvate and toxicology screen

-Neuro assessment

-GCS
-Assess for raised intracranial pressure / herniation
-Assess for focal neurologic disease
-Assess for history or signs of seizures

-If concern for infection, give broad spectrum antibiotics

-Consider LP prior to abx if no delay in doing procedure

-Consider antidotes for specific toxic exposures

-Identify and treat critical elevations in intracranial pressure

-Neutral head position, head of bed 30 degrees, sedation
-Hyperosmolar therapy (Mannitol, 3% NaCl, 7% NaCl)
-Hyperventilation as temporary measure
-Consider intracranial monitoring
-Consider neurosurgical intervention

-Seizure prophylaxis / treatment

-Detailed history and physical

-Head CT

-Consider EEG

-MRI if concerns for stroke

BASIC FLUID THERAPY

OBJECTIVES

- Provision of maintenance requirements
- Replacement of ongoing (excessive/ abnormal) losses
- Correction of deficit/ dehydration
- Volume expansion (mainly shock)

MAINTENANCE REQUIREMENTS

Normal maintenance per 24hrs

Weight	Calculation	Max.
-10 Kg (excludes neonates)	100 mL/Kg	1000 mL/ 10Kg
>10- 20 Kg	+ 50 mL/kg	1500 mL/ 20 Kg
>20 Kg	+ 20 mL/Kg	2400 mL
All patients	1500 mL/m2	

Insensible losses are 400mL/m2/day

Normal maintenance replaces normal urine and insensible losses; since sweat, stool and abnormal losses are normally negligible

Normal maintenance applies provided that:

- Patient is normally hydrated (not dehydrated or oedematous;
 in which case a +ve or –ve 'respectively' balance relative to maintenance is required
- No fluid restriction is necessary eg HF, SIADH, etc
 generally 60-70% of normal maintenance is given
- There are no abnormal losses (radiant warmer, fever, diarrhea, drains, 3rd space, burns, etc)
 measured or estimated non-urinary abnormal losses need to be added to normal maintenance
- Urine output is normal
 in anuric, oliguric or polyuric patients; maintenance shall equal insensible losses + patient's urine output + abnormal losses if present

Amount and type of fluids:

	Condition	Amount	Type
Normal maintenance	Normally hydrated, no restrictions, normal urine & no abnormal losses	1500 mL/m2/day Or check table above	0.2NS[(a)], ≥10% glucose[(b)], 10-20mEq/L K
Restricted maintenance	Oedema, HF, SIADH, etc	Generally 60-70%, higher restrictions may apply	1/3 NS- 0.5NS[(c)], ≥10% glucose, 10-20mEq/L K
Dehydration		Maintenance + deficit	0.5NS- 2/3NS, 2.5-5%[(b)] glucose, 20-40mEq/L K
Insensible loss only	Anuria	400mL/m2, may omit in case of overload	≥10% glucose
Insensible loss + urine output	Oliguria	400mL/m2/day Plus actual patient's urine output per 1,4,12 or 24h [(d)]	As normal maintenance, with/without K
	Polyuria		0.5NS, ≤5%glucose, 20mEq/L K[(e)]
Abnormal losses	Other than in urine	Add amount equal to losses in 1,4,12 or 24hrs[(d)]	Depends on type of fluid lost[(f)]
PD/ CRRT		Consider patient & therapy balance together	

Oral/ enteral fluids, medications, drug infusions, blood products, etc are part of maintenance/ replacement fluids.

Na, K should be monitored and adjusted accordingly. Maintenance potassium should not be added in anuric patients.

(a) In critically-ill children (not small infants), 1/3-0.5NS may be preferable in the first 24hrs

(b) At maintenance rate, use of at least 10-12% glucose is necessary if tolerated, to prevent catabolism and provide some calories. Higher glucose concentrations require central access unless for a very short term. Blood glucose should not consistently exceed 180-200 mg/dL. Higher infusion rates (eg in dehydrated patients) require reducing glucose concentration. Patients with liver cell failure, adrenal insufficiency or metabolic crisis should receive at least 10% glucose, even if insulin is required. Very rapid replacement (eg severly polyuric patients) may not tolerate even 2.5% and glucose-free fluids (± low rate glucose 10% as a separate infusion)

(c) Unless sodium restriction is also necessary (use normal maintenance in this case)

(d) Interval depends on patient's condition and how changing it is. As a start, use 1hr for immediate postcardiac surgery, posttransplantation and those on CRRT; use 12-24h for values close to normal maintenance (i.e. slight increase or decrease) and 4h for most other cases. Increase interval when stable

(e) Extremely polyuric patients are expected to need higher Na and lower K content

(f) For example, upper GI losses by Ringer plus 20 mEq/l potassium, diarrhea by usual rehydration solutions, drains with Ringer lactate, etc

DEHYDRATION & DEFICIT THERAPY

Dehydration	Mild	Moderate	Severe
Description	History of ECF loss, acute wt loss, just detectable dehydration, score 1-2	Significant dehydration: score 3-7, or TWO of (sunken eyes, poor skin turgor, thirst, irritability)	Hypovolemic shock, lethargy* or severe metabolic acidosis* Score 8+ TWO of (sunken eyes, very poor skin turgor, lethargy* or inability to drink*) *of no other cause
Deficit (mL/Kg) 'higher values for smaller patients'	**30-50**	**60-100**	**90-150**

Score allocates 2 points (0=normal, 1=mild, 2=severe) for each of fontanel, tongue, eyes & skin turgor. Two points are added for each of shock, lethargy and metabolic acidosis if present.

Deficit therapy:

Shock should be treated as needed. Patients with severe dehydration who are not shocked may still be given a rapid bolus of 10-20mL/Kg isotonic crystalloid (normal saline) over 30-60 min; which is then subtracted from deficit

There are rapid, slow & very slow methods for correction of dehydration:

Rate	Rapid	Slow	Very slow
Duration of correction	≤ 6hrs	12-24hrs	≥48 hrs
Indications	- rapid fluid losses - acute severe dehydration (eg GE) In children with no concerns from rapid hydration (eg HF, electrolyte disturbance, etc)	Most cases	Concerns of osmotic shifts (eg DKA, hypernatremia)
Technique	Give deficit over 6h (? 3-4h in children >2yrs)	Give 24h maintenance + deficit over 24hrs Other approaches: - Give ½ deficit (+1/3 daily maint.) in 8h, then ½ deficit (+2/3 daily maint) in 16h - Give deficit + (½ daily maint) over 12h	Give maintenance + 40-50 mL/kg/day until corrected (typically will take 2-3 days)

Type of fluid: 0.5NS – 2/3NS with glucose 2.5% (rapid)-5% (slow) and 20 mEq/l potassium (more if needed).

Potassium should not be added in anuric or hyperkalemic patients although hypokalemia may still be corrected (by formula, without maintenance K) in anuric patients if needed.

Dehydration should be reassessed frequently. Severity may be different from initial assessment and patients may have abnormal losses or have/ develop acute kidney injury.

HYPONATREMIA

Hyponatremia denotes a free water excess relative to Na content; however, ECF volume may be increased, normal or decreased

ECF volume	Pathophysiology	Examples	Management strategy
Reduced (hyponatremic dehydration)	Na loss > water loss	GE, diuretics, adrenal insufficiency, salt-losing nephropathies, etc	**Rehydrate with higher Na** Crude: use 2/3NS or NS & modify according to rate of Na rise Accurate: add 10-12 ml/kg hypertonic saline to total daily fluids
Normal	Free water gain	SIADH	**Water restriction (60-70%)** Use 1/3NS – ½NS unless Na restriction is also needed
Increased	Water> salt retention	Congestive HF, hepatic failure, nephrotic syndrome	

-NS, 2/3NS & ½ NS refer to Na content. Appropriate glucose & K should be added
-Correction rate at no more than 0.5 mEq/Kg hourly (12/day), preferably even slower (8/day). Check & modify rate of rise after 6h.
-Severe symptomatic cases may warrant initial partial correction with 5-10 ml/Kg hypertonic saline over 2 hrs (to raise Na 4-8 mEq/L which is adequate to reverse acute symptoms), followed by more gradual correction

HYPERNATREMIA

Hypernatremia denotes a free water deficit relative to Na content; however, ECF volume may be increased, normal or decreased

ECF volume	Pathophysiology	Examples	Management strategy
Reduced (hypernatremic dehydration)	Water loss> Na loss	GE	**Give maintenance + 40mL/Kg/day with 1/3NS- ½ NS** **Modify according to rate of Na drop:** - If too slow: ↑rate or ↓Na content, & vice versa
Normal	Free water loss	DI	**Give 40mL/Kg free water (eg G5%) per 24h and the remaining fluids as usual**
Increased	Hypertonic Na intake	Concentrated formula, sea water, Na bicarbonate, etc	

-½ NS & 1/3NS refer to Na content. Appropriate glucose & K should be added.
-Na content can be given as bicarbonate in acidotic patients (eg 30mL bicarbonate in 500 mL glucose 5% + 5mL KCl will be 60meq/L Na – between 1/3NS & ½ NS)
-Correction rate at no more than 0.5 mEq/Kg hourly (12/day). Check & modify rate of drop after 6h.
-An accurate calculation is possible, based on urine vol, urine Na, insensible loss & current ECF volume status to determine 24h maintenance water & Na requirements separately. Then, allocate 40 mL/kg of the total volume as free water and give the remainder with the proportional amount of calculated Na.
-PD is needed for refractory hyperNa, intractable acidosis or associated AKI with need for dialysis

HYPOKALEMIA

-Interpret K together with pH (a low pH ➔ extracellular shift of K so level will increase; correction will lower K correspondingly)

-Mild hypokalemia which is non-progressive may be corrected by increasing K intake.

Otherwise, correction can be achieved using the formula:

Body weight X deficit (target-actual K) X 0.6 mEq (0.3 mL)

Amount to be diluted in normal saline & given over few hrs (correct 0.5mEq/l per hr).

NEVER GIVE CONCENTRATED KCl.

Target K is 3.0 for chronic hypokalemia & where complete correction is risky or not necessary, 4.0 for those with or at risk for arrhythmia or during correction of acidosis & 3.5 for most cases

HYPERKALEMIA

-Interpret K together with pH (correction of acidosis could correct hyperkalemia)

-K> 7 mEq/L, rising or with ECG changes is an emergency

Emergency measures for hyperkalemia:

(all may be temporary & K can rebound so if hyperkalemia is severe and K cannot be eliminated from the body, dialysis will be needed)

- Calcium gluconate IV to reverse membrane effects of K
- Intracellular shift with bicarbonate, iv or nebulized salbutamol &/or glucose-insulin

Potassium removal can be achieved using loop diuretics, cation exchange resin (Ca or Na polystyrene sulfonate) or dialysis. The administration of corticosteroids & fluids in cases of hyperkalemia secondary to adrenal insufficiency is also effective.

Fluids & mixtures by sodium content

	Na (mEq/L)	Examples	Glucose & K content
Isotonic	±150	Normal saline Ringer & lactated Ringer (Na slightly less)	None K (4-5 mEq/L) in ringer & LR
2/3 NS	±100	Glucose: saline 1:2	1/3rd source glucose (3.3% if using 10%), No K
	80-90	IV rehydration premixed solutions	Glucose <5% K varies by brand (8-30mEq/L)
½ NS	77	Glucose: saline 1:1 Half normal saline	½ source glucose (5% if using 10%, 2.5% if 5%), No K
1/3 NS	±50	 Glucose: saline 2:1 Glucose: ½ NS 1:2 1:19 bicarb: glucose (25mL/500) 1:16 bicarb: glucose (30mL/500)	No K. Using 5%, final glucose: 3.3% (6.7% with 10%) 1.7% 5% 5% & higher Na (60mEq/L)
0.2NS	±30	Glucose 10%: saline 4:1 Glucose 10%: 25%: saline 3:1:1 Neomaint Pedimaint	8%, No K 11%, No K 12%, K 10mEq/L 10%, K 20mEq/L, higher Na (37)
___	0	Glucose (all conc.)	

Kadalex is glucose 5% with 27mEq/L potassium

To increase K by 20 mEq/L add 5mL KCl per 500 mL, or 1 mL/100mL

FORMULAE & CALCULATIONS

Body surface area (m2) = $\frac{\text{weight (kg) x 4 + 7}}{\text{weight (Kg)+90}}$

mL/day to drops/ min: divide by 100

Limit of oliguria:
-400 mL/m2/day
-25% normal maintenance
-1mL/Kg/hr up to 10 Kg, 0.5mL/Kg/hr >10Kg, 20mL/hr in those >40kg

Limit of polyuria:
-2000 mL/1.7m2/d
- 0.8-1.0 x normal maintenance
- 3mL/kg/hr up to 10 Kg

Universal fluid balance formula:
Obligatory intake (medications, infusions, transfusions) + enteral*&parenteral nutrition + IV fluids= Insensible loss + Urine output + Abnormal losses + Ultrafiltration

*only calculate oral/enteral fluids and water content of foods (eg 85% of milk formula)

Metabolic acidosis:
Weight x deficit x 0.33 = mEq NaHCO3 (or mL 8.4% solution)

Deficit= base deficit -5, also ½ base deficit or 15- patient bicarbonate

Hypokalemia:
Weight x deficit x 0.6 = mEq KCl (or 0.3 = mL KCl)

KCl MUST BE DILUTED in normal saline
Deficit= target K – patient K , Target K can be 3-4 meq/L depending on the condition
Correct a deficit of 0.5/hr (eg if patient K2, target 3.5; then deficit =1.5 & min. duration 3hrs)

Hyponatremia:
Rapid correction 5-10 mL/ Kg hypertonic saline in 2hrs

Glucose: insulin for hyperkalemia:
MIX 0.5g/kg glucose (eg 5ml/kg glucose 10%) with 0.1 u/kg soluble insulin & give over ½ hr
May double both, i.e. 1g/kg glucose & 0.2 u/Kg insulin

24-hr infusion calculation:
Basic formula 1.44 mg/Kg/day (or 1.5 mg/25hrs) = 1 mcg/Kg/min
Body weight (Kg) x target dose (mcg/Kg/min.) x 1.44 mg ➔ dilute to 24mL & give at 1 mL/hr

BLOOD COMPONENT THERAPY

- Verify patient and product. Errors can & do occur
- Never leave the 'remainder' of an opened transfusion unit (in a refrigerator or otherwise) for later use
- In case of a suspected transfusion reaction, MANAGE THE PATIENT, obtain a blood sample from the patient as well as from the remaining bag, as well as the bag identification information
- Most PICU transfusion triggers are based on indirect (neonatal &/or adult) evidence

Red cell transfusions: Transfuse 15 mL/Kg of packed RBCs or 20 mL/Kg of whole blood, at 5 mL/Kg/h (unless there is major active bleeding) and complete within 2-4hrs

Transfusion triggers:

- Hypovolemic shock associated with acute blood loss
- Refractory shock and Hb <10g/dL
- 7g/dL is appropriate for stable ICU patients. Consider higher levels in cases of:
 - -Active bleeding or hemolysis (levels expected to decline)
 - -Significant cardiopulmonary disease (tissue hypoxia) as heart failure, respiratory distress, MV with significant FiO2 (>40%), severe sepsis, persistent unexplained tachycardia
 - -Major organ ischemia (eg ischemic stroke)
 - -8 g/dL for otherwise stable patients undergoing surgery, post-operative patients with symptomatic anemia & patients with malignancies

Fresh frozen plasma: Transfuse 12-15 mL/Kg

Indications:

- Bleeding or high risk of bleeding (eg invasive procedure, perioperative, trauma with massive red cell transfusion) associated with coagulopathy due to multiple, undetermined or unavailable factor deficiency

The routine use of FFP to correct laboratory abnormalities in patients with low bleeding risk is NOT indicated

- Emergency reversal of Warfarin anticoagulation (Prothrombin Complex Concentrate preferred if available)
- Anticoagulant protein replacement (antithrombin III, protein C, ..)
- TTP (PE) and aHUS due to possible complement inhibitor deficiency (PE or transfusion)

Platelets: Transfuse 1-2 units per 5-6 Kg weight.

Indications:

- Bleeding or an invasive procedure and PLT <50,000/ cu mm. Higher counts are needed in cases of: -PLT <75,000/ cu mm and major trauma, major hemorrhage or massive transfusion
 - -PLT <100,000/ cu mm for intracranial or inner eye hemorrhage or CNS trauma
- Bleeding or an invasive procedure and significant platelet dysfunction
- Patients at risk of bleeding:
 - -PLT <10,000/ cu mm
 - -PLT <20,000/ cu mm and age ≤3Mo, severe sepsis or other hemostatic abnormalities

In those with thrombocytopenia due to peripheral platelet destruction (eg ITP), PLT are NOT given irrespective of count; except with life-threatening bleeding or an invasive procedure

Apheresis (single donor) units are preferable. AntiD is recommended for Rh –ve girls receiving platelets from Rh +ve donors.

AKI AND RENAL REPLACEMENT IN PICU

- AKI is common in PICU

- AKI ≠ need for dialysis; rise in creatinine &/or abnormal urine output denote AKI

- Despite it's present in AKI classifications, eCCl/ eGFR by formulas based on serum creatinine requires a STEADY-STATE creatinine; which is unusual in AKI !!

- Abnormal urine output generally refers to oliguria/ anuria, although this is not an essential requirement for diagnosing AKI. Inappropriate polyuria is also an abnormal urine output and may be associated with tubular injury or tubulointerstitial nephritis

pRIFLE

	eCCl	Urine output
Risk	Decrease by 25%	<0.5 mL/kg/hour for 8 hours
Injury	Decrease by 50%	<0.5 mL/kg/hour for 16 hour
Failure	Decrease by 75% or <35 mL/min/1.73m^2	<0.3 mL/kg/hour for 24 hour or anuric for 12 hour
Loss	Persistent renal failure > 4weeks	not applicable
End stage	Persistent renal failure >3 months	not applicable

KDIGO

	SCr	Urine output
Stage 1	Increase 1.5-1.9 x baseline or increase ≥27µmol/L	<0.5 mL/kg/hour for 6-12 hour
Stage 2	Increase 2-2.9 x baseline	<0.5 mL/kg/hour for ≥12 hour
Stage 3	Increase >3 x baseline **OR** SCr ≥354 µmol/L **OR** Initiation of RRT **OR** eGFR <35 mL/min/1.73m^2	<0.3 mL/kg/hour for ≥24 hour **OR** anuric for ≥12 hour

Renal angina index (RAI) = Risk of AKI * Signs of injury

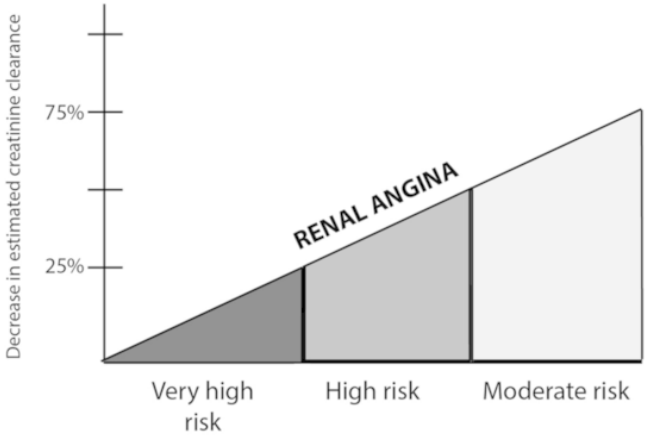
RISK

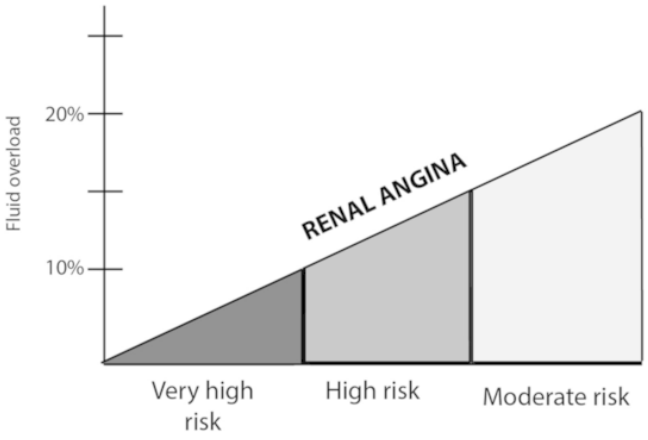
Risk	Score
Moderate	1
High	3
Very High	5

X

INJURY

↓ eCCl	% FO	Score
No change	<5%	1
↓ 0-25%	≥5%	2
↓ 25-50%	≥10%	4
↓ ≥50%	≥15%	8





Very high risk patients

eCCl decrease of 25%

High risk patients

eCCl decrease of 25-50%
OR
Increase in 27 µmol/L SCr over baseline
OR
Fluid overload 10%

Moderate risk patients

eCCl decrease >50%
OR
Fluid overload >15%

AKI: management concepts

- Establish and maintain appropriate renal perfusion (BP, CO & tissue perfusion) and oxygenation.
DO NOT KEEP ANY PATIENT HYPOVOLEMIC AND DO NOT WITHHOLD NECESSARY VASOPRESSORS!!

- Urinary tract obstruction needs to be ruled out quickly

- A challenge with loop diuretic (eg furosemide 2-3 mg/Kg) may be warranted in anuric/ oliguric patients who are not hypovolemic. Higher doses (up to 10 mg/Kg) may be attempted once in anuric patients not responding to standard dose. A constant furosemide infusion of 0.5-1 mg/Kg/h may maintain response in oliguric patients. NEVER MAINTAIN INFUSION >6hrs IN A NON-RESPONSIVE PATIENT.
The use of low dose dopamine to improve renal perfusion is currently NOT recommended

- Manage fluid balance:

-Maintenance = insensible losses + patient urine output + abnormal losses if present.
-Deficit may be added or fluids restricted below maintenance (even not given at all if no concerns eg hypoglycemia) in overloaded patients.
-Maintenance K is not given to anuric/ significantly oliguric patients who are not hypokalemic; hypokalemia may (& should) be corrected
-Dilutional hyponatremia is managed by fluid restriction; only severe symptomatic or resistant cases need hypertonic saline if bicarbonate (contains more Na than hypertonic saline!)

- Significant metabolic acidosis is corrected with NaHCO3. Maintenance bicarbonate (starting at 2 mEq/Kg/day divided q6h) may be needed. *Concerns: volume, Na content, will lower Ca & K*

- Monitor electrolytes and manage disturbances

- Antihypertensives may be needed

- Avoid nephrotoxic agents (unless critically needed), use less toxic alternatives (when possible) and adjust doses of renally-excreted medications

- AKI is not a contraindication for feeding

- Diagnose & manage conditions with specific treatment eg aHUS, RPGN, etc

Indications of RRT:

1.AKI:

-Fluid overload:

*hypervolemia, pulmonary edema
*refractory extravascular overload 15-20%
*potential overload: persistent oliguria with +ve balance if not tolerated eg cardiac condition or obligatory fluid needs such as transfusions, nutritional support, etc

-Severe/ refractory electrolyte imbalance

(mainly hyperkalemia, sometimes extreme hyperphosphatemia, rarely others)

-Metabolic acidosis when correction with bicarbonate is ineffective or not possible
-Clinically significant uremia (eg with bleeding, encephalopathy, etc)
-Very high or rapidly rising creatinine

2.CKD:

-Acute indication (as AKI) in a patient with CKD (on conservative treatment or just diagnosed)
-ESRD

3.Non-renal:

-Severe resistant electrolyte disturbances (dialysis not common in non-renal cases)
-In-born errors of metabolism (esp. with resistant acidosis or hyperammonemia)
-Intoxication with dialyzable drugs/ poisons
-Cytokine removal in severe sepsis (CVVH?)

ACUTE RRT options

Hemodialysis, Peritoneal Dialysis, Continuous RRT or Sustained Low Efficiency daily Dialysis (SLEDD) - each has advantages and disadvantages

Choice is guided by:

- Patient characteristics (disease/symptoms, hemodynamic stability)
- Goals of therapy (fluid removal, electrolyte correction)
- Availability, expertise, and cost

Peritoneal dialysis (PD)

- Fluid removal follows an osmotic gradient, therefore more dependent on concentration of dialysis solution
- Clearance achieved by PD depends on size of molecule, dialysis fluid osmolality, dwell time, and volume of dialysis fluid
- Simple, inexpensive, not equipment-dependent, possible in hemodynamically unstable patients
- Contraindications: defect in peritoneal membrane, abdominal problems

Intermittent hemodialysis

- Extracorporeal exchange of fluid and solute that occurs across an artificial semipermeable membrane between blood and dialysis fluid moving in opposite directions
- Children have to be hemodynamically stable and be able to tolerate interval between dialysis runs. Not suitable for small infants (except with special circuits)
- Precautions are needed to avoid disequilibrium with rapid urea clearance in severely uremic patients
- Vascular access & anticoagulation needed (but sessions can be done with high flow, repeated saline flush & no anticoagulation)
- Moderate needs in terms of equipment, cost & required expertise. But a HD machine & water treatment unit is needed in the ICU or the patient should be fit for transport to the hospital HD unit

Acute peritoneal dialysis worksheet

Preferential indications:

-Small patients -Unstable patients (would not tolerate HD)
-Risk from too rapid correction (urea, Na) -Non-availability of HD or CRRT

Catheter insertion (rigid trocar-catheter):

- Aseptic procedure
- Appropriate sedation &/or local anesthesia
- Bladder should be empty (catheter)
- Site: midline just below umbilicus, aim towards pelvis, near vertical at 60-70 degrees until peritoneum is entered
- Prefilling with saline/ dialysate at 20-40 mL/kg (unless there is ascites) using needle or wide-bore cannula facilitates entry and reduces injury
- Skin incision (do not widen and do not puncture peritoneum with scalpel, to avoid leak)
- All catheter side holes must be intraperitoneal; that means REMOVE EXCESS HOLES

Roughly keep ½ holes for a child using an adult catheter, for a neonate use ½ child (=¼ adult) holes.
Catheter must be cleanly cut (irregular tip ➔ injury) and any cut portion must be fixed at the back of the trocar (total length on trocar must be preserved) such that the TROCAR TIP SHALL NOT EXTEND BEYOND THE CATHETER TIP AT ALL TIMES.

- Once peritoneum is punctured, proceed with catheter

either pull trocar back few mm so it is within the catheter before advancing OR push catheter over trocar as with a peripheral cannula

- Confirm inflow and outflow, fix catheter and empty the abdomen to start dialysis

(when there is tense ascites, empty gradually to avoid splanchnic shock)

Acute PD standard prescription:

- **Run volume: 40 (30-50) mL/ Kg**
 Rounded to a fraction of the bag (1. ½, 1/3, ¼ L, etc), except patients <5Kg
 Provided it is tolerated (abdominal distension), lower vol. reduces efficiency
- **Dwell time 15-30 min.**
 Shorter time increases ultrafiltration & longer time in case of hypernatremia
- **Calculate balance (in & out) per bag (or max 3-4 runs)**
 Consider patient's total balance (fluid intake, non-dialytic & insensible loss)
 Compare with fluid targets and hydration state
 This is dynamic, REASSESS FREQUENTLY
- **Dialysate composition: standard 1.7% glucose**
 - Increase glucose to increase ultrafiltration (4.25% or add 100 mL D25% per liter)
 - Add heparin 250-500 U/L: 5000 IU ampoule in 10 mL➔ 0.5-1 mL/L
 - Add gentamycin 8 mg/L: 80 mg ampoule in 10 mL➔ 1 mL/L
 - Potassium (2 mL=4 mEq PER LITER) added if/ when potassium is normal
- Laboratory follow up max. after 10-12 runs
- Peritoneal fluid cell count and culture must be obtained if peritonitis is suspected and preferably for all cases after 48h of dialysis
- The max. duration for an acute PD catheter is 48 hrs. *If longer dialysis is needed, options are intermittent PD, catheter change, HD or an extended-duration PD catheter*
-

CRRT:

Proposed benefits:

- Reduces hemodynamic instability
- Excellent fluid control
- Effective control of uremia, electrolytes & acidosis
- Allows for improved nutritional support
- Management of sepsis/ cytokine clearance (high volume CVVH)

Disadvantages:

Need for equipment, high cost, high experience needed. Vascular access & anticoagulation (there are solutions eg regional citrate anticoagulation)

CRRT modalities

Continuous veno-venous hemofiltration (CVVH)

- Provides solute removal by convection, effective for middle molecules (cytokines)
- Uses replacement fluid

Continuous veno-venous hemodialysis (CVVHD)

- Provides solute clearance by diffusion (small molecules)
- Uses dialysate fluid

Continuous veno-venous hemodiafiltration (CVVHDF)

- Provides solute removal by both convection and diffusion
- Uses both replacement fluids and dialysate fluids

Slow continuous ultrafiltration (SCUF)

Just fluid removal; NO dialysate, NO replacement fluid, NO solute removal (other than in the ultrafiltrate; minimal)

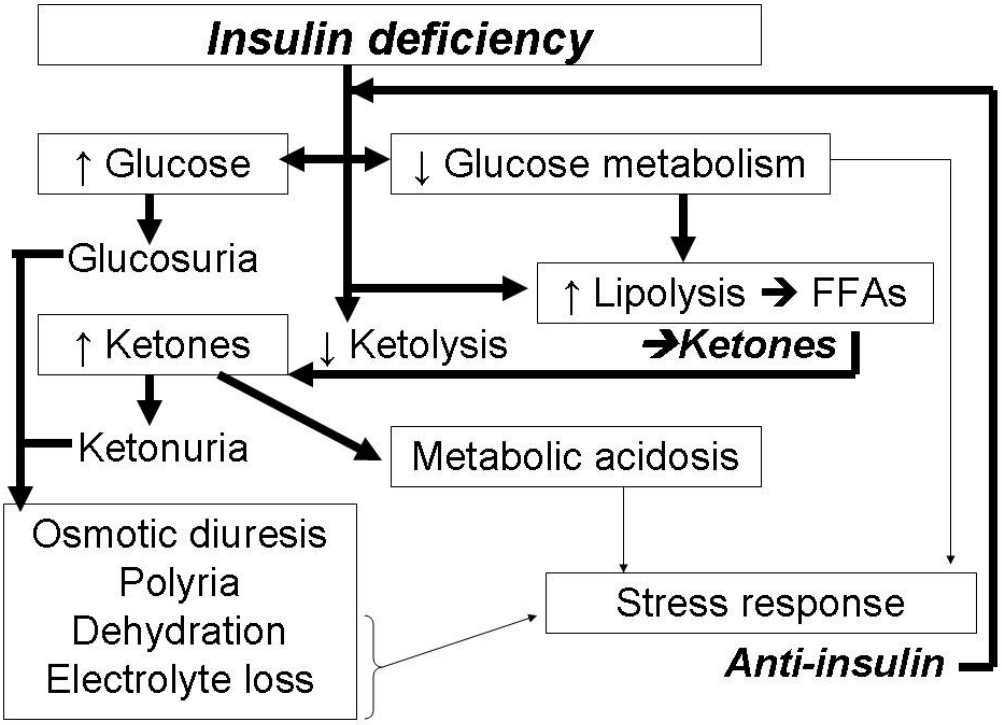
Sustained low efficiency daily dialysis (SLEDD)

The application of slow & extended (but not continuous) hemodialysis with ultrafiltration, on a daily basis, using standard HD equipment
Therapy may replace CRRT in some cases if HD is available at the ICU

DIABETIC KETOACIDOSIS (DKA)

Acute metabolic derangement resulting from failure of insulin-dependent glucose metabolism

Pathophysiology:



The main underlying defect is insulin deficiency, resulting in hyperglycemia & impaired tissue glucose utilisation, with increased FA metabolism & ketone production. Ketones cannot be metabolized because of deficient insulin & carbohydrate metabolism. Glucose & ketones are increased in plasma and excreted in urine, resulting in osmoting diuresis with fluid loss. Ketones lead to metabolic acidosis. Dehydration, acidosis & impaired glucose metabolism lead to increased release of stress hormones which have anti-insulin effects, exacerbating the process.

Precipitating factors:

Infections
Insulin under dosage
Trauma: physical or psychological

Clinical Picture:

-Suspect in known diabetic child who appears sick and/or has any of the precipitating factors *but DKA can be the 1st presentation of diabetes*
- Preceding symptoms: Weight loss - polyuria – polydipsia – vomiting – abdominal pain
- Clinical triad:
 - Dehydration
 - Rapid deep breathing (acidosis), acetone odor
 - Confusion or lethargy, may be coma

Diagnosis:

-Hyperglycemia (>200 mg/dL)
-Metabolic acidosis (pH<7.3, bicarbonate <15 mEq/L)
- +ve urine for ketones
Plasma ketones are elevated but test is not essential for diagnosis

MANAGEMENT:

1- Resuscitation:

-ABC, obtain IV access
-10 mL/Kg normal saline in 30-60 min. for those with severe dehydration or severe DKA (pH<7.1 or bicarbonate <5mEq/L)
-Correct shock if present

2- Laboratory tests & monitoring:

-Clinical monitoring
-Hourly capillary blood glucose (strips)
-Obtain plasma glucose, Na, K, blood gases and urinary ketones and repeat every 2-4 hrs
-Check renal functions, P, CBC and sepsis screen if suspected

3- Fluids:

-Give maintenance + deficit over up to 48 hrs (50 mL/Kg/ day + maintenance)
-Use normal saline. 0.45% saline is used in hyperosmolar cases.
-K is added 30-40 mEq/L after the resuscitation, after the first hour and after the patient passes urine provided K is not elevated.
Initial K may be normal, low or high, but insulin infusion will lower K. *Correct hypokalemia immediately*
-Glucose is added when blood glucose decreases to 250 mg/dL
-Correct hypophosphatemia if present

4- Insulin:

-Started after resuscitation
-Continuous infusion of 0.1 U/kg/hr regular insulin. NO BOLUSES
-Infusion must be continued until correction of ketoacidosis. Glucose is added to fluids when blood glucose reaches 250 mg/dL to maintain glucose >200 mg/dL with insulin infusion
-When acidosis is corrected, and the patient can tolerate feeding, S/C insulin is given then IV is stopped
-Resistant acidosis: suspect sepsis or renal failure

The use of bicarbonate for correction of acidosis in DKA is generally not recommended except in exceptional circumstances. Because:
-Most cases will be corrected by fluids & insulin
-Bicarbonate can exacerbate hyperosmolarity and electrolyte disturbance

5- Management of complications:

- Shock: hypovolemic (polyuria, vomiting & dehydration) or septic
- Brain edema from rapid correction of hyperosmolarity & hypovolemia and from cerebral ischemic hypoxia (shock)
- Pulmonary edema from hyperosmolarity and HF (2ry to acidosis)
- Cardiac arrhythmias (2ry to acidosis & electrolyte disturbance esp. K)

ACUTE HEPATIC FAILURE

Definition

Sudden failure of the liver to perform its functions, namely the metabolic, excretory and detoxifying functions.

It is very serious with bad prognosis and high mortality

Causes

Infections: Viral hepatitis: Hepatitis A, B, D, others, EBV, CMV
Metabolic: Wilson's disease, Tyrosinemia
Toxins / Drugs: Paracetamol, NSAID, Erythromycin, isoniazid, Halothane
Autoimmune: Autoimmune hepatitis
Reye syndrome: Aspirin + Varicella or Influenza *OK*

Pathophysiology

1-Disturbed hepatic metabolic functions

Hypoalbuminemia
Hypoglycemia
Decrease synthesis of coagulation factors
Retention of some amino acids & some short chain fatty acids
Acid-base disturbance: metabolic acidosis
Electrolyte disturbances: hyponatremia & hypokalemia

2-Disturbed hepatic excretory functions

Elevated serum bilirubin (Except in Reye syndrome)
Elevated serum bile salts

3-Disturbed hepatic detoxifying functions

Elevated blood ammonia level

Encephalopathy results mainly from elevated ammonia, also some amino acids (eg methionine, glutamine) and short chain fatty acids. Other factors contributing to cerebral oedema include vasodilatation, hypoalbuminemia and hyponatremia

Clinical Picture

- Progressive Jaundice (Except in Reye syndrome)
- Bleeding tendency, GIT bleeding, bruising
- Persistent vomiting
- Disturbed level of consciousness (Hepatic encephalopathy)
- Cerebral edema
- Secondary bacterial infections
- Renal failure (Hepatorenal syndrome)
- Respiratory failure

Laboratory findings

- Elevated serum transferases: AST (sGOT) & ALT (sGPT)
- Elevated serum bilirubin (Except in early stage & in Reye syndrome)
- Prolonged prothrombin time (PT)
- Hypoglycemia
- Elevated blood ammonia
- Low serum albumin
- Others: acid-base & electrolyte disturbances

Management

Close Monitoring

Clinical Monitoring

- HR, RR, BP, Temperature
- Bleeding
- Urine output, edema, ascites
- Level of consciousness (LOC)
- Signs of brain edema
- Evidence of infections

Laboratory Monitoring

- Liver functions
- Blood ammonia
- Blood sugar
- Kidney functions
- Prothrombin time (PT)
- Serum electrolytes

Conservative Measures

1- Control of water, electrolyte and glucose homeostasis
IV fluids, adequate glucose. Fluid restriction to 60-70% of requirements may be needed for edema/ ascites ± diuretics
IV Salt-free albumin
2- Feeding
Oral/ Nasogastric/ TPN
Low protein intake, lactulose & neomycin to reduce ammonia and other toxin production
3- Control of bleeding:
Correct coagulopathy: fresh frozen plasma, IV Vitamin K
Fresh blood transfusion
Antacids
4- Treatment of brain edema
Head elevation
Fluid restriction, correct serum albumin and Na, IV Mannitol
Therapeutic hyperventilation
5- Control of infections
Antibiotics (guided by cultures and consider hepatic toxicity)

Drastic Measures Charcoal hemoperfusion, Liver transplantation

ACUTE PRESENTATION OF IN-BORN ERRORS OF METABOLISM

- In-born errors of metabolism can present with an acute metabolic crisis that can be fatal
- Individual in-born errors of metabolism are generally rare; however, taken collectively they are not that rare

When to suspect?

- Consanguinity or previous unexplained sibling deaths (most are autosomal recessive)
- Symptoms may be triggered by a minor illness
 - Early non-specific manifestations:
 - Lethargy, poor feeding, vomiting
 - Tachypnea
 - Neurological signs eg hypotonia, abnormal movements, convulsions
 - Can rapidly progress with a sepsis-like or encephalitis-like picture and MOSF
- Deterioration despite apparently adequate therapy

Management

A. Basic investigations:

- Blood glucose (hypoglycemia may be present)
- Blood gases, anion gap, lactate and ketones (ketoacidosis, lactic acidosis)
- Plasma ammonia may be elevated
- Liver function tests
- Others according to systems affected

B. Specific diagnostic tests:

- Obtain samples for extended metabolic screening

C. Treatment of the acute episode:

- Monitoring & system support
- No nitrogen intake initially. Adequate calories & glucose ± insulin infusion to prevent catabolism
- Correct metabolic acidosis with Na bicarbonate
- Medical treatment for hyperammonemia
- Some disorders are vitamin-responsive eg biotin, pyridoxine
- Dialysis for failure of conservative measures especially with refractory acidosis or hyperammonemia

D. Diagnosis & management of the specific disorder

APPENDIX

Vital signs

	Normal HR	Normal RR	Hypotension (SBP)	Normal BP	
				Systolic	Diastolic
0-2 Mo	100-180	30-60	< 60 (term)	60–90	20–60
2-12 Mo	80-160	30–50	< 70	87–105	53–66
1-2 Y	80-130	24–40	< 70 + (age in years x 2)	95–105	53–66
Preschooler	70-120	22–40		95–110	56–70
School age	60-110	18–30		97–112	57–71
Adolescent	55-100	12–20	**< 90**	112–128	66–80

50th percentile systolic blood pressure= 90 mmHg + (2 x age in years)

Emergency drugs

Drug	Dose (IV)	Comment
Adenosine	0.1 mg/Kg (Max dose: 6 mg) IV	Give rapidly No effect in 2 min: 0.2–0.3 mg/Kg (Max dose: 12mg)
Albuterol (Salbutamol)	MDI: 4–8 puffs q 20 min with spacer or through ETT Neb: 0.15 mg/Kg (max 5mg) 2.5 mg (<20 Kg) and 5 mg (> 20 Kg) q 20 min Continuous Neb: 0.5 mg/Kg/hr IV: 5-10mcg/Kg (max. 0.5mg) slow bolus Infusion: starting 1mcg/kg/min (<10Kg), 10mcg/min (>10Kg); max.2mcg/Kg/min (to 40mcg/min)	Asthma, Anaphylaxis, Hyperkalemia
Amiodarone	5 mg/Kg IV/IO*	Give rapid bolus for VF/pulseless VT Give over 20–60 min for SVT and ventricular dysrhythmias
Atropine	0.02 mg/Kg IV (may give ETT)	Min dose 0.1 mg Max dose: 0.5 mg (child) 1 mg (adolescent)
Calcium Gluconate 10%	50-100 mg/Kg IV	Give slowly (Max. rate 200 mg/min) Max dose: 2 g/dose Calcium chloride dose is 1/3 calcium gluconate
Dexamethasone	0.25-0.6 mg/Kg PO/IM/IV	Max dose: 16 mg
Epinephrine (Anaphylaxis)	IM: 0.01 mg/Kg (0.01 ml/Kg) 1:1,000	Max dose: 0.3 mg or 0.3 ml

Epinephrine (Code)	First dose IV/IO*: 0.01–0.03 mg/Kg 1:10,000 concentration Second & subsequent doses (asystole) 0.1 mg/Kg 1:10,000 or 1:1,000 ETT: 0.1 mg/Kg 1:1,000 concentration	Repeat as needed per PALS protocol for code Only use 1:10,000 concentration for neonates
Epinephrine (croup)	0.25–0.5 ml racemic solution (2.25%) in 3 ml saline via inhalation	Up to 3 ml of 1:1000 epinephrine mixed with 3 ml saline via inhalation is alternative if racemic not available
Flumazenil	0.01 mg/Kg IV (Max dose: 0.2 mg)	Give over 15 seconds May repeat at 1 minute intervals up to 4 times Max total dose: 0.05 mg/ Kg to 1 mg
Furosemide	0.5–1 mg/Kg IV	Higher doses may be needed in patients with renal impairment & those with pulmonary oedema/ hypertensive crisis
Hydralazine	0.1-0.5 mg/Kg	Hypertensive emergency/ urgency Titrate to effect, max. dose 20 mg
Hydrocortisone (stress dose)	2 mg/Kg (50-100 mg/m2) IV bolus	Adrenal insufficiency, catecholamine refractory shock
Hydrocortisone (anti-inflammatory)	5 mg/Kg	
Insulin (regular)	0.05–0.1 Units/Kg/hr infusion	Diabetic Ketoacidosis (adjust rate to response)
Lidocaine 2%	1 mg/Kg IV/IO*	May repeat dose in 10–20 min.

Magnesium Sulfate	25 mg/Kg IV/IO*	Give over 10–20 min; faster in Torsades de Pointes Max dose: 2 g
Methylprednisolone (antiinflammatory)	2 mg/Kg IV/IO/IM	Status Asthmaticus, Anaphylaxis Max dose: 60 mg
Methlprednisolone (high dose pulse)	Up to 30 mg/Kg	Infuse over 1 h Max dose 1 g
Naloxone	0.01 mg/Kg IV/ET	Titrate to effect Max dose: 2 mg
Sodium Bicarbonate	1-2 mEq/Kg IV	Empirical dose, when accurate correction by formula not used Give over 15-30 min. Use 1 mEq/ml for > 6 months Use 0.5 mEq/ml for < 6 months

Antiepileptics

Drug	Dose (IV)	Comment
Levetiracetam (Keppra)	50 mg/Kg load over 15 minutes Maintenance: (divided q12h) <6 Mo: 14-42 mg/Kg/d 6Mo-3y: 20-50 mg/Kg/d >3y: 20-60 mg/Kg/d	Max dose: 2500 mg Max. 3g/day
Phenobarbital	15–20 mg/Kg load at 1 mg/Kg/min Maintenance: (divided q12h) 3-8 mg/Kg/d	May cause hypotension Max. total loading 30 mg/Kg Up to 1 g max
Phenytoin	15–20 mg/Kg load at 1 mg/Kg/min Maintenance: (divided q12h) 3-8 mg/Kg/d	Max. total loading 25 mg/Kg Up to 1 g max.
Valproate	20-60 mg/kg/day Divided q8hrs	

Sedation

Drug	Dose (IV)	Comment
Fentanyl	1-2 mcg/Kg/dose IV q 30–60 minutes Continuous: 1-2 mcg/Kg/hr	Rigid chest with rapid administration
Ketamine	0.5-2 mg/Kg/dose IV (consider with asthma)	Bronchodilator IM dose 4-5 mg/Kg
Midazolam	Sedation: 0.05-0.1 mg/Kg (max. 2mg) Deep sedation/ seizures: 0.1-0.2 mg/Kg (max. 5mg) Titrate q2-3 min., max cumulative dose: 0.6 mg/Kg (<6y), 0.4 mg/Kg (6y+), 10 mg Continuous: 1-6 mcg/Kg/min	Avoid in hemodynamically unstable patient
Morphine	0.1– 0.15 mg/Kg IV q 3–4 hours Continuous: 10 –30 mcg/Kg/hr	Ventilated patients may receive larger doses, more frequent intervals Avoid in asthma patients
Propofol	Bolus: 1-4 mg/Kg SHORT TERM ONLY infusion 25–100 mcg/Kg/min	Avoid in hemodynamically unstable patient
Thiopental	2-5 mg/Kg IV Continuous: 1-3 mg/Kg/hr	Avoid in hemodynamically unstable patient

Neuromuscular blocking agents

Ventilation support required. Patient must be sedated

Drug	Dose (IV)
Succinylcholine	1 mg/Kg/dose (induction/ rapid sequence intubation)
Atracurium	0.3-0.6 mg/Kg/dose (duration 30-60 min) Infusion: 0.3-0.6 mg/Kg/h
Pancuronium	0.05-0.1 mg/Kg/dose (duration 2-4 hrs)

Continuous infusions

Drug	Dose (IV)
Alprostadil (PGE1)	0.01–0.1 mcg/Kg/min 0.02–0.05 mcg/Kg/min typical range
Amiodarone	10 mg/Kg/day for 4–5 days then 5 mg/Kg/day
Dobutamine	5–20 mcg/Kg/min
Dopamine	Dopaminergic: 1-4 mcg/Kg/min B adrenergic: 5-10 mcg/Kg/min Vasopressor (alpha): 10-20 mcg/Kg/min
Epinephrine**	0.01–1 mcg/Kg/min 0.01–0.5 mcg/Kg/min typical range
Esmolol	Load: 500 mcg/Kg Maintenance: 50–1000 mcg/Kg/min
Isoproterenol	0.05–2 mcg/Kg/min 0.1–0.5 mcg/Kg/min typical range
Lidocaine	Load: 1 mg/Kg Maintenance: 10–50 mcg/Kg/min
Milrinone	Load: 50–75 mcg/Kg IV over 10 minutes 0.25–0.75 mcg/Kg/min
Nitroglycerin	0.5–10 mcg/Kg/min 0.5–5 mcg/Kg/min typical range
Nitroprusside	0.5–10 mcg/Kg/min 0.5–2 mcg/Kg/min typical range
Norepinephrine**	0.01–2 mcg/Kg/min 0.01–0.5 mcg/Kg/min typical range
Vasopressin**	Cardiovascular support: 0.05–5 milli–units/Kg/min 0.1–0.5 milli–units/Kg/min typical range Diabetes Insipidus dose: 1–10 milli–units/Kg/hr

** Central line preferred, *** Central line preferred, but short duration peripheral IV acceptable

APPENDIX II: LOGISTIC ISSUES

INDICATIONS OF ADMISSION

Patients with acute potentially reversible (including acute on top of chronic) conditions associated with:

- Multiple Organ System Failure

- Respiratory failure needing mechanical ventilation
- Upper airway obstruction needing intubation
- Advanced or increasing respiratory distress or developing respiratory exhaustion (impending respiratory failure or possible need for mechanical ventilation)

- Unstable cardiac arrhythmias
- Advanced shock, shock refractory to volume expansion, or shock requiring inotropic/ vasopressor support
- Pulmonary oedema or refractory heart failure; needing inotropic support

- Refractory status epilepticus
- Need for therapeutic mechanical ventilation for refractory increased intracranial pressure, refractory pulmonary hypertensive crisis or refractory hypercyanotic spells
- Guillian-Barre Syndrome with rapid ascent or bulbar affection

- Deep coma, rapidly deteriorating consciousness
- Hypertensive emergencies
- Severe acute metabolic derangements; such as intractable metabolic acidosis, refractory or severe hyperkalemia, complicated DKA, acute metabolic crisis, etc
- Advanced hepatocellular failure
- Severe Stevens Johnson syndrome

***Need for continuous monitoring, difficult or complex fluid/ electrolyte management or intensive care-dependent procedures*

GENERAL RULES

- All admissions are subject to availability of places
- Critically ill patients with immediate absolute indications (those in bold) who had already been admitted to an in-patient ward of one of Cairo University Pediatric hospitals shall have absolute priority
- Patients who can be effectively managed in wards/ intermediate care units as appropriate shall have a lower admission priority
- Neonates should be admitted to the NICU
- Patients smaller than 5 Kg are to be admitted on servos
- Specific isolation requirements are to be considered.
- *Cases of sepsis who have meningitis as part of sepsis and are not suspected of having meningococcal disease or have completed 24 hrs of appropriate antimicrobial therapy may be admitted*
- Otherwise indicated cases who do/might need renal replacement therapy shall be admitted normally and then co-managed with nephrology as appropriate.
- *Primary renal patients (on renal replacement or with intrinsic acute or chronic renal disease) who are critically ill and fulfill admission criteria for nephrology ICU should be admitted there so long places are available there*
- Routine post-operative intensive care should be managed in surgical units so long places are available there

MEDICAL RECORDS

Some of the standards related to Medical Records (JCI)

*** Information about the patient's care and response to care is communicated among medical, nursing, and other care providers during each staffing shift, and between shifts.**

*** The patient's record(s) is available to the care providers to facilitate the communication of essential information.**

*** Information related to the patient's care is transferred with the patient.**

*** The organization uses standardized diagnosis codes, procedure codes, symbols, abbreviations and definitions.**

*** The organization initiates and maintains a clinical record for every patient assessed or treated.** The clinical record contains sufficient information to

Objectives of medical records:

- ***identify the patient***
- ***support the diagnosis***
- ***justify the treatment***
- ***document the course and results of treatment***
- ***promote continuity of care among health care providers.***

The clinical record of every patient receiving emergency care includes
the time of arrival, the conclusions at termination of treatment, the patient's condition at discharge, and follow-up care instructions.

Organization policy identifies those authorized to make entries in the patient clinical record and determines the record's content and format.

Every patient clinical record entry identifies its author and when the entry was made in the record.

As part of its performance improvement activities, the organization regularly assesses patient clinical record content and the completeness of patient clinical records.

CONSULTATION, URGENT CONSULTATION and COMANAGEMENT

Consultation is an essential part of providing healthcare to patients with multiple morbidities and to patients whose treatment involves different specialties/ subspecialties. Defining the relationship (consultation versus co-management) between different physicians is important for coordinated delivery of healthcare to inpatients.

Although frequently termed so, seeking the opinion of a senior staff member of the same unit and specialty where the patient is admitted is strictly NOT a consultation.

The default situation is that the unit where the patient is and the physicians responsible for that unit are primarily responsible and ultimately accountable for the patient.

CONSULTATION:

A consultation means you are seeking the opinion of someone, generally from another specialty or subspecialty. You will provide info on pt background, your management, why are you consulting and patient progress on current management if available.

Consultations should generally be processed during working hours and consultant is expected to respond within 24 hrs. Consultations or follow-up consultations requested in the afternoon for non-urgent matters (eg new lab results, afternoon rounds, etc) should NOT be processed as urgent.

Response is consultant opinion (assessment and recommendations) regarding stated condition. He isn't expected to do anything directly to the patient other than assessment, unless you agree together based on his opinion. Recommendations are NOT binding and it's YOU who generates or modifies your patient's management plan. Patient is always yours and your responsibility.

You are responsible for arranging a follow-up consultation if needed, unless consultant specifically tells you he'll do it.

URGENT CONSULTATION:

The same except that expected response is within 1 hr provided consulted service has an emergency on call system. Consultant may initiate initial emergency management provided it's not interfering with your ongoing plan.

Following the urgent response, YOU are responsible for arranging a follow-up consultation if needed, unless consultant specifically tells you he'll do it.

COMANAGEMENT:

Another service, specialty or subspecialty is managing a patient admitted to your service with you, not instead of you. Both parties must approve this. A service referring a patient to an ICU is NOT automatically a request for comanagement, but the request may be made with the referral.

Once approved, comanagement means they need to follow the pt without you asking. That means they have to come daily without request, unless this is not required by the extent they are involved in comanagement AND this has been made clear at the previous visit.

Each party must discuss their management plans with the other at each follow-up visit and this works BOTH WAYS. The patient must have ONE integrated management plan and this must be coordinated by the primary physician responsible. For all PICU admissions, all decisions related to system support are primarily the unit's, not the comanaging service's, responsibility. Urgent matters within the scope of a comanaging service shall be notified to them as would be any urgent matter for their own patient.

NOTES

- All requests and responses to consultations must be documented, dated and timed
- All comanaging physician actions and notes must be recorded as progress notes
- Physicians processing consultations/ comanagements are NOT expected or authorized to communicate with parents regarding assessments, interpretations or decisions that have not been approved by the unit. For comanagements, ongoing communication with parents may be required and will thus need an agreed upon plan and communication approach between parties

PICU ROUND

CLINICAL EXPECTATIONS:

- **Be on time**
- **You should know your patient better than anyone else**
 - Full assessment of new cases
 - Be aware of what actions are to be followed up (labs, etc).
 - Know the results of studies performed.
 - Know about significant afternoon and overnight events
 - If there is a procedure to be done, even though you may not be the one performing it, you should be involved.
 - If there are pertinent social situations please update team
- **Prepare (preround)**
 - Check the current orders
 - Check current respiratory support
 - Check current infusions, rates & doses
 - Check monitors, vitals, fluid balance and past 24 hrs highs & lows
 - EXAMINE THE PATIENT
- **Presentations are concise, clear, with relevant information presented in orderly fashion**
- **Know about the disease processes you are treating**
- **Wash your hands!!!**

ROUND OUTLINE:

- **Who's the patient**; briefly (basic data, time & reason for admission, past, present and new problems, other significant issues)
 A past problem is one preceding the present illness, a present problem has been present on admission while a new problem has occurred during admission
- **What happened yesterday**
 (events, changes, new results compared to previous state, etc)
- **Brief report on each of current**:
 - Respiratory support & assessment
 - Vascular access, infusions, fluids, CV status
 - Pharmacy
 - Nutrition
 - Consultations, comanagements
- **Situation by system**
 (previous plan ➔ response ➔ needs)
- **Additional concerns** (team, family, social, legal, etc)
- **Updated plan** and orders/ implementation needs

NOTE:

- DOCUMENT; you will forget
- Critical medications/ infusions must be calculated by two people
- Revise all calculations at least daily, don't just copy
- Inform nurses about new plans, and changes during the day as soon as possible
- Follow your plans, change when necessary

Referral, transfer and discharge

- Where is your patient going?
- How would he go there?
- Who should care and who will follow?
- Did they receive everything necessary for that? Ensure continuity of care
- Is all YOUR paperwork complete?

TRANSFER NEEDS

Level	Condition	Individual responsible
1	-Hemodynamically stable -Airway/ resp. stable -No need for PPV -No need for continuous monitoring -No or maintenance IV that does NOT require titration -Physician intervention (including new orders) unlikely	Patient attendant
2	-Patient needs monitoring, medication &/or IV drip modification -Stable, no need for PPV -Physician intervention (including new orders) unlikely	House officer or qualified nurse
3	-On BVM ventilation with stable airway, or with ETT secured -Stable, physician intervention unlikely	Brief (few minutes): -Resident; or -House officer or qualified nurse experienced in respiratory therapy Significant: Resident
4	-Hemodynamically unstable; -Airway/ resp unstable; or -Possible need for physician intervention; including new/ changing orders	Resident

Patient transport

- **First, why?** What exactly will be done and Benefit vs risk
- **Second, when?** Ok you must prepare your team, your patient and the receiving unit/ service. But also, is NOW a good time or better next morning?
- **Third, how?** Means of transport, what infusions, equipment supplies and most importantly personnel are needed? Not only consider procedure success but also patient safety and emergency responsiveness
- **Check everything before you leave:**

YOUR PATIENT, communicate with other party, complete paperwork, support needed, oxygen and equipment, anything required from unit by the other party, do you need consents/ approvals?

TIPS FOR COMMUNICATION WITH PARENTS

PRINCIPLES

-Use the *"I'm OK, you're OK"* mindset
-*Talk WITH parents.* NOT to patients, NOT at parents. It is a TWO-WAY process; Listen
-Don't communicate in a setting or way that may convey arrogance or ill care
-Respect their dignity, privacy and information confidentiality
-Parents want to know the truth, but you need to be supportive, non-judgmental and empathetic
-*The transaction is between the patient and the hospital.* Parents represent the patient and you represent the hospital. NEVER act as though you, or your unit, is something separate from somewhere else in the hospital (the general ward, ER, surgeons, lab, management, etc)

PROCESS

-Understand their concerns and needs, and elicit relevant information.
-Explain the child's illness and its treatment. The explanation should be clear, complete, and in a language that the parents can easily understand.
-Check understanding
-Avoid information gaps and misconceptions
-Respect autonomy. Treatment options should be explained clearly and completely, so that they can make informed decisions.
-Convince parents of their child's situation and/ or needs when necessary and respond to issues such as prior impressions, denial, unwarranted fears, cultural issues, etc where they jeopardize correct informed decisions

KEYPOINTS

-All patients in ICUs are critical, when they are not they won't be with us. Critical means they need support and many bad events may occur. We're here to follow these and respond. That's why your child is in the ICU
-Exact expectations are not common in the ICU. People respond very differently.

PROBLEMS

-Never show that you don't know anything that parents know about the case; which happened after admission or was previously told to one of your colleagues. *That means: know the case before talking to parents*
-Responses that base information availability or (worse) patient management on non-existing factors should be avoided at all cost (eg an investigation to be done outside, an unavailable medication, a delayed report, the previous resident, the surgeon, a non-present consultant, etc)
-Discuss complications and unfavorable outcomes appropriately as possible outcomes of disease and its management
Their main valid concern will be that everything necessary has been and will be done. Address this appropriately
-Respect parents' natural responses to unfavorable outcomes and don't take them personal. Do NOT unnecessarily assume being attacked and react by opposing parents
- Blaming others is never a good practice unless you are in position of officially judging them
-If parents blame someone or otherwise give a constructive feedback or concern,
do not automatically act against them but show respect for the concern, do not admit an error for now and report the feedback to be checked. Exceptionally however, if the concern is clearly illogic, misinformed, exaggerated, of minimal impact or otherwise inadmissible, clarify this confidently but without giving the sense of attack.

Do
• Greet the child and parent by name. • Smile. • Sit down when talking. • Try to talk in the patient's language. • Direct the conversation to relevant directions. • At the end of the consultation, ask if the parents have any questions. • Engage the parents in a dialogue. • Give time for the parents to absorb and understand the content of your explanations, then to ask questions.

Don't
• Look at your watch frequently. • Appear to be in a hurry. • Use too many medical terms. • Talk with your hand on the door handle, or foot outside the door. • Interrupt all the time. • Start examination and then write out a prescription before the main problem has been identified. • Give long lectures as explanation. • Ignore concerns mentioned by parents.

(Partly abstracted from Mehta PN. **Communication Skills – Talking to Parents. Indian Pediatr. 2008; 45:300-304)**